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A.M.A. ARCHIVES OF

# OTOLARYNGOLOGY

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# A. M. A.
# ARCHIVES OF OTOLARYNGOLOGY

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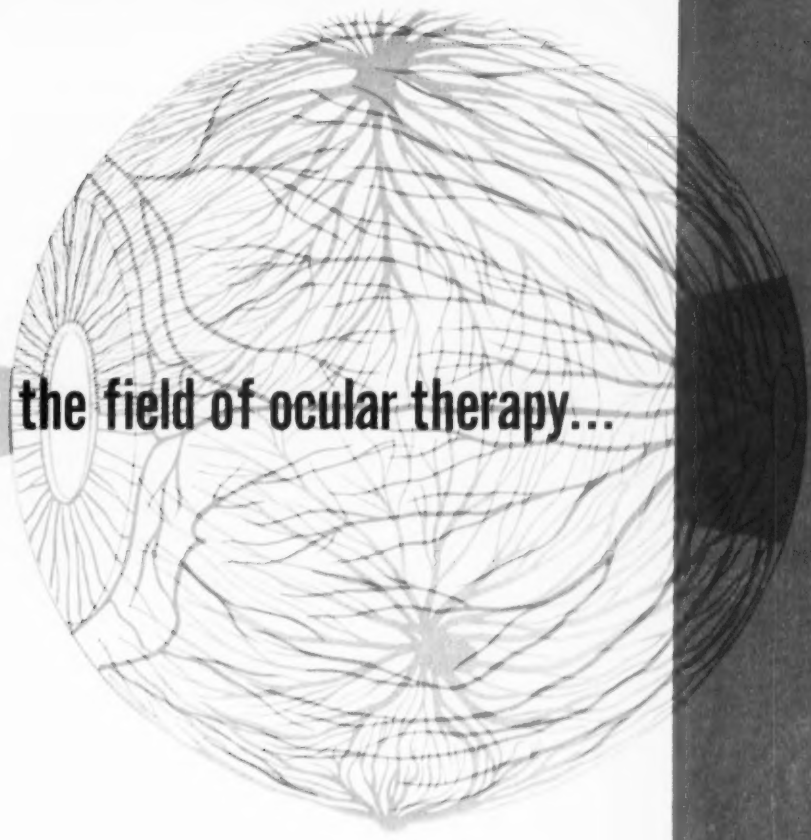

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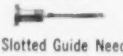

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Editorials

# Sixth International Congress of Otolaryngology

From May 5 to May 10 the Sixth International Congress of Otolaryngology was held in Washington, D. C., under the eminent leadership of Arthur Proetz, St. Louis, President, and Paul Holinger, Chicago, Secretary-General, with the assistance of a committee composed of prominent American otolaryngologists. The Congress was attended by some two thousand delegates from nearly every country in the world. There were two hundred scientific papers read and discussed, seventeen scientific exhibits, television shows of surgical operations and techniques, elaborate moving picture, or cinema, programs, and a host of commercial exhibits. The Sixth Congress was most successful, and was even blessed by good weather.

The First Congress was held in Copenhagen, Denmark, in 1928, under the presidency of Schmiegelow, with subsequent ones in Madrid, Berlin, London, and Amsterdam, this being the first in America, although in 1912 there was held in Boston the Ninth International Congress of Otology under the presidency of C. J. Blake, of Harvard.

When we consider the great number of titles presented and discussed, the reports of research and of new surgical and therapeutic measures advanced, it is evident that the term applied to otolaryngology, "a dying specialty," is entirely unjustified. Otolaryngology is a most vigorous and expanding specialty, which deals with more diversified subspecialties than those concerned with organs or areas in other parts of the body. Many of these subspecialties were pioneered by otolaryngologists, such as maxillofacial surgery, rhinoplasty and otoplasty, allergy, some types of neurosurgery, speech and voice therapy, audiology, the modern temporal-bone surgery, bronchoesophagology, and diagnosis of brain lesions by the aid of vestibular reactions. The otologist is taking an important part in industrial medicine in the study of the prevention and amelioration of occupational deafness.

Truly this Congress has disclosed that for a "dying specialty" the patient is exhibiting a most vigorous response to otolaryngological therapy.

Following the Washington meeting, the Sixth International Congress of Bronchoesophagology took place in Philadelphia on May 12 and 13; an International Congress on Audiology in St. Louis, May 14 and 15, and an International Voice Conference in Chicago, May 21 and 22.

GEORGE M. COATES

# Cramps of the Professional Voice

**JUAN ANDRADE PRADILLO, M.D., Mexico, D. F.**

A painful, paroxysmal, although temporary, muscular contracture is a typical characteristic of the voice professional's cramp affecting fibers, a single muscle or various muscles in the larynx, or others normally aiding in the formation of the voice. There is also temporary aphonia.

In 1950, when writing on vocal fatigue, I expressed my opinion that cramps might occur similarly to athletic cramps.[1] Jean Tarneaud [2] described them for the first time in 1955.

Cramps may affect not only extrinsic as well as intrinsic muscles of the larynx, but also other muscles forming a part of the vocal organs, as in the thorax, abdomen, pharynx, mouth, etc.

The extrinsic muscles of the larynx make it go up, down, forward, or in a bascule manner.

The intrinsic muscles include the tensors, the dilators, and the constrictors. We shall make special mention of the tyroarytenoids, commonly called vocal chords. These muscles are formed by two types of fibers, arychordals and tyrochordals, which are not parallel to the free edge of the vocal chords. All the muscular fibers are inserted like a comb's tooth on the free edge. When the fibers receive an actuating potential they become shortened, and in doing so the free edge of the vocal chord is shaped into a concavity toward the internal portion. When the nervous flux ceases, the chords relax, returning to the original form because of their elastic nature. The fibers resemble those of the myocardium from the point of view of their histologic constitution as well as their physiology, since they have the most interesting property of fibrillating, of moving rhythmically, of vibrating. The recurrent action is similar to that of the cardiac branches of the vagus.[3-6]

This muscle of the larynx has therefore the activity not merely of contracting like most muscles but also of vibrating, thus requiring a proportionally greater quantity of nutrients and especially oxygen to satisfy the metabolic requirements that develop with its functional activity. In this aspect it can be compared to the cardiac muscle, which vibrates in a similar fashion and also requires greater quantities of oxygen.

The etiology and pathogenesis of vocal cramps do not differ from those of other cramp cases such as writer's, swimmer's, nocturnal cramps, etc.

By studying the frequency of these cramps we are led to believe that it is quite considerable, although it is difficult to study a sufficient number of cases, since normally the patients do not identify the symptoms with a cramp but disregard them as pertaining to "trac" (vocal anxiety). Furthermore, many of these patients will hide their condition, considering that it may be caused by nervousness or a poor vocal technique.

It has been necessary to interview many voice professionals, such as singers, speakers, announcers, singing teachers, and jai alai booking agents to obtain a certain amount of information, which cannot as yet be considered sufficient.

It may be inferred that these cramps are more frequent after the age of 40.

Vocal fatigue is a determining factor. The cramps occur after a prolonged period of vocal exercise. The cause is that in the muscular metabolism during the anaerobic phase the carbohydrates pass through vari-

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ous transformations: glycogen—glucose—glucose-6-phosphate — pyruvic acid — lactic acid; and an exaggerated muscular activity can promote the formation of an excessive quantity of pyruvic acid, thereby disturbing the succeeding phases of the metabolism. The incomplete combustion originated, in turn, creates a lack of oxygen (oxygen debt) necessary for the proper functioning of muscular fibers, especially when the activity is intense.

This anoxia, like cramp cases in other parts of the body, may be caused by arterial disturbances since they provoke a defective blood flooding. However, the arterial alterations might be recognized in other vascular locations and in patients of a more advanced age, but this cause is considered as exceptional.

Fundamentally, as has been mentioned, the determining cause of this condition is fatigue, leading to anoxia and metabolic upsets. Fatigue might easily take place, for instance, when an artist is rehearsing a number of different works at the same time, having to adapt his voice to each one of them and undergoing excessive strain.

It has been said that pain may be produced by a compression of the sensitive nervous filaments; this view is not acceptable; the pain must be attributed to the local anoxia since it is quite comparable to the angina pectoris pain.

Apart from the main factor, which is fatigue, some special body conditions are necessary:

Metabolic changes brought about by hormonal diseases and by those of general character, disturbances of glucide metabolism, and uricemia, as pointed out by others, all have a bearing in the production of cramps.

In this connection, as well, there are hypotheses based on some facts.

Electromyograms of cramped muscles have been obtained, and they are similar to those obtained when there is calcium deficiency in the nerves.

A decrease of calcium in the blood stream provokes tentany, a spontaneous and deranged activity of the motor nerve cells followed by a permanent contracture of the muscles. Calcium metabolism is subject to the action of certain hormones. Calcium elimination is accelerated by thyrotoxin, and, conversely, the parathyroid glands secrete another hormone acting as a buffer to maintain a constant concentration of calcium in the blood.

On the other hand, some suprarenal corticosteroids and thyrotoxin have the property of increasing the sodium necessary for nervous activity. This explains why cramps may develop as a consequence of sodium losses after the use of diuretics, or from excessive diaphoresis.

Inflammatory processes and the irritation centers affected by reflex mechanisms have been mentioned as causes of muscular cramps. The psychoneurotic factor has an influence on voice professionals as in writer's cramp. I attribute this condition to the fact that the psychoneurotic will adopt a poor posture, will make unnecessary contractions, and will talk fast with no rhythm or relaxation, thereby producing undue fatigue.[7-12]

Even though the etiology and pathogenesis of cramps are unknown, it can be assumed that anoxia plays an important role.

## Clinical Considerations

After vocal fatigue, produced by the causes already mentioned, an intense pain develops, generally on the sides of the larynx, making it impossible for the person to emit any sound. It rarely lasts over one minute. The patients, trying massage as a corrective, may be able to go on singing, although the cramp may return immediately or after a few days when the patient tries vocal exercise. Afterwards, cramps may return sporadically.

Vocal cramps should not be confused with other phonetic impediments such as "trac," neurovasomotor monochorditis, coup de fouet, "crac," etc.

The pathologic "trac," or vocal anguish, is determined by an emotional state. Besides the phonetic disturbances which may lead to the incapability of emitting some sounds, there are general and psychic phenomena, such as pallor, cold sweats, dryness in the mouth, and a sensation of ill-feeling leading to depression. These conditions are due to an adrenalin discharge which accompanies all emotional states. The "trac" belongs to a disturbance of the neurovegetative life caused by the anxiety factor.

In the "crac" there is a hypercontraction of the glottis, and sounds do not come out.

In neurovasomotor monochorditis the reddening of a single vocal chord is present. Speech shows only slight modifications, but singing is almost impossible; it cannot be done for a long period, and there is fatigue. It may result from a vasomotor dystonia, from functional strain, or from laryngeal trauma of vocal origin.

In the coup de fouet of Moure there is a submucous hemorrhage in one of the chords, produced by a great glottic effort. Intense pain is present, as well as aphonia followed by dysphonia, which may become incurable.[13-16]

## Cases Observed

Among the cases studied there are the jai-alai book makers. They are in general strong and large men who in their youth were actual players. It is interesting to hear them shout, placing bets between spectators and raising their voices way above the noise and yelling of an emotional public that watches the game and oftentimes gambles small fortunes. They work about six hours per day. They normally use their powerful baritone voices, although a few, to avoid fatigue, use a biphase or falsetto voice.

I examined 20 cases and found that 25% suffered from vocal cramps.

All the cases observed, including singers, teachers, speakers, radio and television announcers, were carefully studied according to the methods of Tarneaud and Garde.

Case 1.—Woman 70 years of age, Mexican, 47 years in the singing profession. She is a contralto with a magnificient voice even in its last years. Her voice span is from E flat up to C-3. The quantity of pulmonary air was measured with a spirometer at 4.8 liters. She is a diabetic with up to 480 mg. of glucose in the blood and at one time had a coma. During that period she had to prepare several works of different nature, adapting her voice to each one of them. This caused her fatigue, and on three occasions she had aphonia without dyspnea, with intense pain in the sternocleidomastoid extending to the left side of the larynx and lasting about one minute. She also experienced cramps in the left hand, in spite of being righthanded, because while concentrating on her work she would have involuntary contractions in her left hand. She showed some hyperactivity in the suprarenals. Puberty was precocious, at age 9½ years, and menopause at 36.

Comments: The cramps in her vocal organs, like those of her left hand, were caused by fatigue. Disturbances in glucide metabolism, as well as the psychic factor producing unnecessary contractions, and sometimes the adoption of poor posture, were causes that had an influence in the production of those cramps.

Case 2.—Woman 38 years old, Mexican, shorthand teacher. She gives dictation to her pupils about eight hours daily, in a loud voice, as fast as possible, and almost without rhythm or modulation. She had chronic tonsillitis with toxic manifestations, because of which she underwent operation. With excessive vocal fatigue, she experiences intense pain in the left side of the larynx and aphonia without dyspnea, disappearing within in one to three minutes. Her voice is correct.

Comments: This cramp of the vocal organs is produced by fatigue. Possibly the infectious state of the tonsils was a factor, since the cramps have not recurred after the operation, although she was advised to use a better vocal technique.

Case 3.—Man 46 years old, Spaniard-Basque. He was a jai-alai player and for eight years he has acted as a jai-alai booking agent. His voice is a dull baritone when he speaks but strong and clear when he talks loudly. He works using his voice about six hours daily. He suffers from frequent hoarseness, had tonsillitis, and was operated on. When he reaches a state of vocal fatigue he feels pain in the middle portion of the sternocleidomastoid, with aphonia as well. This lasts for about one minute, although at times a mild pain persists for several days. He has also had the same pain during the night, severe enough to awaken him. Cramps in the abdominal

muscles have also appeared at times of great vocal strain. On one occasion he also experienced a spasm of the larynx with dyspnea. He has also had nocturnal cramps in the legs. His vocal chords are thick and red, and their vibration is noted to be irregular when seen with a stroboscope. He has a deflected nasal septum with a small orifice, and dryness of the mucous membrane.

Comments: This man has made poor use of his vocal organs, exerting extreme effort. Furthermore, his vocal technique is not correct, as is shown by the reddening and poor vibration pattern of the vocal chords. This has caused the cramps of the sternocleidomastoids and, I believe, also of the intrinsic laryngeal, muscles, since aphonia has been present. Even though no alteration in other organs is apparent, there are without doubt conditions which account for the abdominal cramps and others such as his nocturnal cramps in the legs.

CASE 4.—Man 46 years old; Spaniard from Andalusia. Since the age of 6 years he has acted as a singer in zarzuelas. For the last four years he has been a jai-alai booking agent. His voice is baritone. Oftentimes at night he has awakened, feeling intense pain in the middle portion of the sternocleidomastoids, which recedes in about one minute when he moves his neck about. He has not tried speaking at those times. His normal voice is dull, but it is clear and strong when loud. Fatigue sets in very markedly when he becomes angry; his voice pitch changes, and dysarthria occurs. The vocal chords are thick, and there is secretion. Defective vibration is seen with the stroboscope. He suffered leg cramps when he was an athlete and he now has them after sitting for several hours playing cards.

Comments: The cramps of the vocal organs may have been caused by fatigue, as were those in the legs when he exercised. Those present when playing cards had a static cause, poor posture.

CASE 5.—Man 65 years old, Mexican, in the singing profession for 40 years, baritone. He has had a powerful and clear voice with 2 octaves of extension. He has diabetes, occasionally with 30 gm. of sugar in the urine and 150 mg. in the blood. In 1920 he had dysphonia because of vocal fatigue and had to reeducate his voice. During that time he suffered intense pain in the central portion of the sternocleidomastoid and in the left side of the larynx. He was unable to emit any sound. There was no dyspnea, and the pain subsided after about one minute with the application of massage. After a five-minute rest he would be able to sing again, although at times the pain and contracture would repeat. Sometimes he would have to stop singing altogether, and the same clinical symptoms would appear again the next day if he tried singing. During that period he had an excess of vocal work. He has suffered nocturnal cramps in the legs and arches of the feet.

Comments: This is a typical case of cramps in the vocal organs caused by fatigue with disturbances of the glucide metabolism resulting in nocturnal cramps in the feet.

CASE 6.—Woman 43 years of age, Mexican, singer of popular songs, works for three hours once a week and for one hour all other days; soprano. Her voice is strong and clear; the pitch is accurate and homogeneous. She has suffered from nasal polyps and has been operated on several times on this account. She does not vocalize and rehearses only little with the orchestra; therefore she has to exert her voice unduly in order to sing. This causes fatigue and intense pain in the right side of the larynx, with complete loss of voice. This is followed by coughing, but after a few moments she is again able to sing. It has not been unusual for this situation to be repeated after a few days. O. R. L. examination revealed nothing unusual.

Comments: Undoubtedly this is a case of cramps in muscles of the vocal organs due to fatigue.

CASE 7.—Man of 56, Spaniard from Toledo. He was a jai-alai player and is now a booking agent. He works six hours daily with his voice. Baritone, with a strong voice, good extension and clear pitch. When working very hard he has felt pain in the sternocleidomastoid and has had almost complete aphonia, being able to speak only in a very low voice. The pain lasts for one or two minutes, disappearing after he gesticulates and moves his neck about. When he was a jai-alai player he suffered from cramps in the legs caused by fatigue. He has traces of tonsils which were poorly operated on.

Comments: The cramps in the sternocleidomastoid were caused by fatigue, with a possible influence of the tonsil remnants. Cramps in the legs were also caused by fatigue.

CASE 8.—Man 47 years old; Spaniard—Basque. He was once a jai-alia player but for the last 15 years has been a booking agent. He suffers from chronic tonsillitis. Works six hours daily and tires frequently. He has suffered from slight prelaryngeal pain and aphonia with a duration of about one minute. In such cases the same clinical

picture would return, if he tried to speak loudly, and it would be necessary for him to decrease the intensity of his voice to keep on talking. He has suffered from cramps in the legs and arms due to fatigue.

Comments: The cramps of the vocal chords, like those of the legs and arms, were produced by fatigue and possibly influenced by the tonsil infection.

Case 9.—Man 29 years old, Spaniard. He is a professional soccer and jai-alai player as well as jai alai booking agent. He works five hours daily using his voice, which is a strong baritone with clear pitch.

Frequently he has a slight prelaryngeal pain and aphonia, which last not more than one minute. These symptoms are repeated exactly when the patient raises his voice, and he has been able to speak again on decreasing the intensity of his voice.

## General Comment

It is noticeable that the majority of persons suffering from vocal cramps also experience cramps in other muscles of the body.

In all cases fatigue has been the determining factor.

The symptomatology of voice cramps is similar to that of other cramps: pain and incapability for a short time of carrying out the normal function of the muscles affected.

In persons who suffer from voice cramps, one finds alterations in glucide metabolism, as well as psychoneurotic factors which cause the adoption of incorrect postures, which in turn produce fatigue; diaphoresis also occurs after strenuous exercise. All of this is found in persons affected by other cramps, anoxia being the possible result of all these upsets.

Upon investigation it can be demonstrated that the frequency of vocal cramps, like that of other types, is quite considerable.

## Conclusions

1. The voice professional's cramp does exist.

2. The etiology and pathogenesis are no different from those in other cramps. It is believed that anoxia plays an important role.

3. The frequency is surely greater than is generally believed.

4. The clinical picture consists essentially of pain in one side of the larynx with incapability of phonation, rarely lasting more than one minute.

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# Nose and Nasality

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The term nasality, derived from Latin nasus (nose), seems to serve excellently the purpose of characterizing certain acoustic impressions of abnormal speech. Yet science also asks for a definition of a term. Now either the term nasality suggests a very unclear definition, namely, "something abnormal going on in the nose," or the term proves to be wrong after an exact definition of the condition has been found.

Observation shows that there are fundamentally two entirely different acoustic impressions that indicate "something going on in the nose." Furthermore, normal speech also sometimes produces the impression of "something going on in the nose." The latter fact also could suggest the use of the term "nasality," and certainly the same term should not be used for normal and abnormal conditions. The impression of "something going on in the nose" in normal speech can, if well defined, serve as a starting point for understanding the abnormal acoustic findings, inaccurately called "nasality." The physiologic features suggesting the term nasality are the peculiar sounds which characterize the nose sounds, m, n, ng. Exact phonetic examinations have revealed that the vowels neighboring a nose sound change slightly toward the peculiar sound characteristic for nose sounds (B. von Dantzig[1]). Nasality (normal and abnormal) can be heard, seen, and felt. To see it one puts a laryngoscope under the nostrils while the examinee says m or n or ng. The laryngoscope should not be placed under the nostrils before the patient says the sound, for the "nasality" produces a film of moisture on the laryngoscope. Such a film will also result from nose breathing. Therefore, if the laryngoscope is put under the nostrils before the examinee speaks a nasal sound, the breathing moisture will hinder the "nasality" moisture from being visible. A phonetic graphic method, also leading to visible findings, will be dealt with later in this paper.

The tactile method consists of putting slightly (without pressure) the tip of the physician's middle finger at one ala of the nose of the examinee. Vibrations will be felt during the pronunciation of a nose sound. All these tests, and one more which will be described later, can be used also for pathological conditions.

"Nasality" is, as said before, a physiologic finding, and therefore the term should not be used for pathological findings. Already a theoretic consideration makes it probable that "something going on in the nose" may impress the listener as pathologic if it is either too much or too little as compared with the physiologic feature. This theoretical consideration really coincides with the facts. There are cases in which the peculiar acoustic characteristic of the nose sounds shows in all the voiced sounds. In the vowels it becomes most obvious. This condition has been named open nasality, or rhinolalia aperta. These terms also seem to me not to help the student in his endeavor to understand the pathologic procedures that lead to the acoustic impression. For "open" (aperta) is exactly what gives the physiologic nose sounds their peculiarity. The soft palate (which rises in all other speech sounds of English and, touching the posterior wall of the pharynx at Passavant's pad, closes off the mouth from the nose*), hangs down,

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* According to Hajek,[2] also lateral muscles of the pharynx approach the rising velum.

thus giving the air the opportunity to enter the nose. Therefore, in my opinion, the adjective "open" (aperta) should not be used to signify a pathologic condition. I have introduced the term hyperrhinolalia (too much rhinolalia) for the speech impediments in which all the voice sounds carry with them the sound normally characteristic only for the nose sounds. Hyporhinolalia (too little rhinolalia), on the other hand, should signify those cases in which the nose sounds show too little or are missing altogether.

Hyperrhinolalia may be organic or functional. The organic condition may be congenital or acquired. Congenital forms are cleft palate, holes in the palate or too short a velum (which if lifted cannot reach the posterior wall of the pharynx) or a paralyzed velum. Acquired hyperrhinolalia results from traumatically acquired holes in the palate, from destructive diseases, like syphilis, from scar formation after tonsillectomy or adenoidectomy. Acquired but functional hyperrhinolalia can derive from pains after these operations. In the latter case the patient develops the habit of not lifting or of lifting insufficiently the velum, even after the pains have gone (habitual form, according to H. Gutzmann[3]). The cases of postoperative hyperrhinolalia are of special interest to the rhinologist. In a paper devoted to this speech trouble[4] I pointed out that, although it is very probable that hyperrhinolalia can be due to surgery, it has not been proved so far. Only if the existence of a preoperative hyperrhinolalia can be denied, is one entitled to speak of postoperative hyperrhinolalia. Exact logopedic examinations before tonsillectomy and before adenoidectomy are missing. The mirror test, described above could have revealed an insufficient function of the velum, while acoustically hyperrhinolalia was not evident, at least not to the, in this respect, untrained ears. Very large tonsils may have to a great extent blocked the passage of air into the nose. Adenoids (also enlarged posterior ends of the turbinates) may have damped the hyperrhinolalia. Here I want to cite a diagnostic method which is, as hinted above, especially good for hyperrhinolalia. It is Gutzmann's[3] ah-ee test. While the patient says ah-ee several times, the physician alternately closes both nostrils with his thumb and index finger and lets them open again. In this way (provided air passes the nose) while closing the nose he produces a damping influence upon the hyperrhinolalic tone in the nose. The difference between the damped and the undamped tones becomes very conspicuous. Such a difference, of course, will not show in normal speakers because air does not pass the nose during pronunciation of the vowels. An especially exact diagnostic method has been elaborated by Luchsinger,[5] who uses the tone-frequency spectrometer to write the tones of which hyperrhinolalia consists. After removal of the tonsils and/or adenoids, the hyperrhinolalia will become much more evident. The rhinologist should not be surprised by such an event. If a weakness of the velum shows by exact examination before operation, the surgeon may possibly revise his indication for an operation. If there is no other choice but to operate, he should warn the patient, or, in the case of a child, the parents, that hyperrhinolalia will become obvious and that will ask for logopedic treatment. This, of course, holds also for patients with other anatomic abnormalities in the palate, such as holes or clefts.

Functional hyperrhinolalia may be, as mentioned above, due to surgery (pains). Imitation is another source of this trouble. I have seen cases with a functional hyperrhinolalia who had one parent or older siblings with an organic or functional form of hyperrhinolalia. As to the cases whose speech trouble cannot be traced back to either of the two sources (pain, imitation), we are forced to assume that the speech centers just lack "the correct idea." Sometimes psychogenic reasons may be found—a subconscious resistance of the patient toward normal speech.

The neurologic differential diagnosis between organic and functional paralysis or paresis of the velum can be performed by pressing the tongue down violently with a

tongue depressor. A nonorganically affected velum will rise, while the organic form of paralysis and paresis, respectively, will become evident by a lack of this reaction or a weak response.

Weiss[6] has described "active hyperrhinolalia," a speech trouble in which the velum, not being paralyzed, is drawn downwards by contractions of the palatopharyngeal and palatoglossal muscles. I recommend making the differential diagnosis between the paralytic (paretic) and active form in the following way: If the patient sitting on a sofa pronounces a long-lasting "Ah" and slowly during the continuous pronunciation lies down, in the paralytic (paretic) form the hyperrhinolalia will gradually diminish because the velum will gradually approach the posterior wall of the pharynx, obeying gravity. In the active form no such change will take place. The differential diagnosis is, of course, important for the two forms of hyperrhinolalia and for their diametrically different treatments. The methods will be described later.

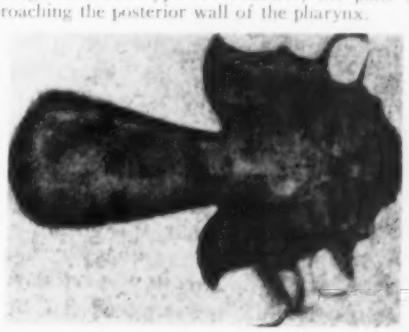
Now, the hyporhinolalia should be dealt with. It also may be organic or functional. An avenue toward the understanding of the organic form has already been opened in the discussion of the damping influence of swellings in the nasopharynx and/or in the nose in hyperrhinolalia. If an abnormal amount of air does not enter the nose—in other words, if no hyperrhinolalia exists—the damping influence will be noticeable in nose sounds and the neighboring vowels. In this second form of "nasality"—I here use the wrong term again, for it is the one used if the insight into the essence of "something going on in the nose" is insufficient—of course all the tests for hyperrhinolalia will fail.

The functional form of hyporhinolalia consists of the velum overworking. In nose sounds it either rises too much, so that not enough air can enter the nose, or it prevents all air from entering by touching the pharyngeal walls, or, finally, it gives the air only too short a chance to enter, so that the nose sound is too short and the neighboring vowels do not have any rhinolalic component.

The treatment of the organic form of hyperhinolalia is surgical† or orthodontic. For the latter procedure, as far as it is prosthetic, I can refer to some findings which might interest the rhinologist. In a case of total cleft palate I plugged the meatuses with oil-soaked cotton in order to find where the real seat of hyperrhinolalia was. Although the rest of the nose was still open for the air coming from the mouth, the hyperrhinolalia disappeared. That proves that hyperrhinolalia is localized, chiefly at least, in the meatuses. With these findings a peculiar fact can be explained, namely, that some cases with cleft palate or a paralyzed velum show no or a very weak hyperrhinolalia. Those are patients with swollen posterior ends of the turbinates which prevent the air, although it comes into the nose, from entering the meatus. On the basis of those findings, Dr. Schalit and I have constructed the so-called meatus obturator. The other types of obturators are designed to hinder the air from entering the nose by covering the clefts or holes of the hard palate and by substituting for the missing or cleft or too short velum a plate approaching the posterior wall of the pharynx (Fig. 1). It is easily possible to make up in this way for the defect of the hard palate. But since the normal velum is mobile and by its

†The methods, like building a flap of the pharyngeal muscles to elongate a short velum (Rosenthal operation) cannot be dealt with in this paper.

Fig. 1.—Older type of obturator, the plate approaching the posterior wall of the pharynx.



mobility fulfills its speech function, it seems to be impossible to have an immobile plate substitute for it. As a matter of fact, if the substituting plate is too long, normal rhinolalia (m, n, ng) will be wanting; if it is too short, hyperrhinolalia will persist. Warneckros tried to use the pharyngeal muscles which protrude in normal speech when the velum rises (chiefly Passavant's pad), but unfortunately in many cases of cleft palate these muscles are underdeveloped. Attempts were made to design a mobile plate to substitute for the velum. Between the plate covering the hard palate and the plate substituting for the velum a hinge was inserted. In order to lift the plate, a muscle-bridge had to be built from the remnants of the soft palate. The hinge was placed upon this muscular bridge. The small amount of muscle was, as a rule, not strong enough to lift the plate, and so the device never became popular.

The meatus obturator works on quite a different principle than do all the older forms (Fig. 2). A plate is built, covering the hard palate (from below) and protruding about 0.3 cm. beyond the posterior border of the hard palate. On the protruding portion a softened compound is placed and pressed with one finger up into the nasopharynx and forward toward the posterior nostrils until it covers all of the nostrils. The patient, asked to say "m" or "mama," will, of course, show a complete hyporhinolalia. Then a hole is drilled through the compound in the middle. This hole should be enlarged until hyporhinolalia has disappeared, and, since the air cannot enter the meatuses, there will be no hyperrhinolalia. Fortunately, the nose sounds can still be spoken. They probably are formed in the still open middle of the nose.[7-9] (Most of the cleft palate cases do not articulate all the consonants correctly. Therefore logopedic training is necessary before and after the meatus obturator has been constructed.)

Fig. 2.—Meatus obturator. The vertical frame rising into the nasopharynx closes the entrances into the meatuses.

For the treatment of paralyzed or paretic or slightly too short velum we use the so-called pushing exercises. First described by me in 1932, they were published in English[9,10] in 1933 and 1941.

In case of sudden voluntary contraction of one group of muscles, other muscle groups tend to contract, reinforcing the function of the first group (as in grimacing when lifting a heavy weight, clenching the fists during defecation, etc.). The pushing exercises[11] consist of simultaneous movements of the arms and phonation. The patient is first instructed to raise his fists to his chest with elbows close to the body and to push his arms down in one quick, elastic sweep. During the downward swing of the arms, the fingers should not open. When the arm movements are completed, the palms should land at the front of the thigh.

As soon as this can be done without undue tension in the shoulders and upper arms, the patient is requested to say "Ah" at the very moment he starts pushing his arms down. Other vowel sounds are used alternately. Then one syllable is spoken with pushing and afterwards repeated with the idea of pushing in mind. Then single words are used, and later the patient makes one pushing at the first syllable and speaks several words, maintaining in his mind the attitude of pushing. The results are very satisfying.

In functional hyporhinolalia we use the so-called chewing method.[12,13] Since one can chew food and talk at the same time, and since the nerves, the muscles, and even the

center in the anterior central convolution are the same, the chewing and speaking (articulation) movements must be the same. Since I came to this conclusion (1934) several x-ray films have proved it to be correct. If the patient is taught to think of chewing while speaking, there is no overcontraction of all the muscles concerned in speech (and voice). In this way the overcontractions of the velum in cases of functional hyporhinolalia have disappeared.

Finally, a rare condition should be mentioned, the so-called mixed rhinolalia in which moments of hyperrhinolalia alternate with moments of hyporhinolalia. The reason seems to be that in the presence of "cushions" in the nasopharynx and/or nose the velum at times hangs down so completely that the cushions cannot do the damping sufficiently to extinguish hyperrhinolalia, while in other moments the velum rises so far that the amount of air can be made up for by the damping influence of the cushions. Logically, pushing exercises should first be used to find out whether a sufficient rising of the velum can be achieved to eliminate hyperrhinolalia. If constant or frequent hyporhinolalia should persist, surgery has to follow.

The rhinologist might have received the impression that this paper has led him far away from his professional interests. I hope that deeper consideration will prove to him that, since the nose plays such an important part in speech, the rhinologist's scope is wider than he might think.

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# Repair of the Facial Nerve in Traumatic Facial Palsies

*Results of Decompression, Nerve Suture, and Nerve Grafting in One Hundred Twenty-Seven Cases*

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A traumatic facial palsy is one of the most serious hardships which may befall a patient. The palsy does not endanger life, it is true, but the psychic and economic consequences are so great that suicide is often attempted to bring an end to the suffering.

All our endeavors must be directed toward restoring mimicry. The symmetry and synchronism of the emotional movements of the face are dependent on the close cooperation of the cortical facial centers of both hemispheres, and the only way to reestablish this cortical interplay in cases of peripheral facial palsy is to repair the injured nerve at the site of the lesion.

Previously neurotization and, especially, an anastomosis operation between the facial nerve and another cranial nerve were used in order to reinnervate the facial muscles. By these methods the muscles may eventually acquire sufficient tonus so that the palsy is not noted with the face at rest, and the patient may even be able to perform some voluntary movements. But after procedures mimicry will never return (Fig. 1).

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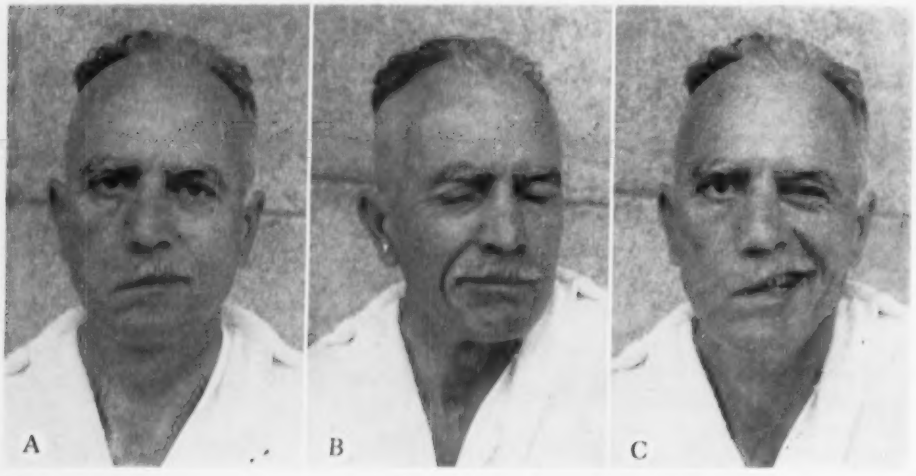
## Methods

To attain our goal, the restoration of mimicry, three procedures are at our disposal: (1) decompression of the facial nerve in the Fallopian canal, (2) nerve grafting, and (3) nerve suture.

Alt (1908) was the first to advocate direct exposure of the nerve trunk at the site of the lesion; Ney in 1922 published a method

Fig. 1.—In this case an anastomosis between the facial and the spinal accessory nerve was performed on the right side six years ago. *A*, face at rest. *B*, voluntary movements when he was told to close the eye and draw the corner of the mouth upwards. *C*, when he was asked to smile or laugh, the movements at the right side were completely lacking. This illustrates that whenever possible the facial nerve should be repaired at the site of lesion. Mimicry will never return after an anastomosis operation.



of repairing the facial nerve intratemporally as worked out on the cadaver, but Bunnell (1927) was the first to perform a successful intratemporal nerve grafting as well as a nerve suture. He was followed by Martin and Smith (1931). Most of the credit for the enormous advances in this field, however, must be given to Ballance and Duel (1932), who experimentally created the foundation on which modern facial nerve surgery rests.

Numerous reports of successful operations from all over the world are now available (and a comprehensive bibliography is included in one of my previous papers), but special attention should be paid to papers by Botman, Botman and Jongkees, Cawthorne, Collier, Findlay, Lathrop, Martin, Maxwell, Sullivan, and Tickle. I have published in a series of papers the results obtained in 300 consecutive cases operated on according to the method of Ballance-Duel and Bunnell. Those dealing with the problems discussed in this paper are cited below.

1. *Decompression of the Facial Nerve.*—Excellent descriptions of the technique have been published by Lathrop, Sullivan, Bunnell, and Maxwell, which should be referred to for detailed information.

In decompression, the Fallopian canal is opened until the site of injury is exposed. In some cases, the continuity of the nerve is unbroken and the palsy may be due to a hemorrhage or an edema, the pressure of which thus is relieved. It may also be due to tearing of the nerve, to a bone spicule accidentally pressed into the nerve sheath during an operation, or to a fragment resulting from a fracture of the temporal bone.

In other cases the continuity of the nerve is destroyed, and then decompression is the first step in performing nerve grafting or nerve suture.

2. *Nerve Grafting.*—If the continuity of the nerve is interrupted the gap may be bridged by means of a nerve graft. Any sensory nerve of the right caliber may be used, and many have been suggested. Bunnell and Boyes have suggested the use of "cable grafts" instead of a single graft of a larger caliber, maintaining that the latter is more inclined to necrotize as compared with several grafts of small caliber. Personally, I prefer a single graft, the distal end of which, if necessary in extratemporal repair, can be split and sutured to the distal ramifications after the division at the pes anserinus. I always use the ilioinguinal nerve, as suggested by Bergström, because it is easy to find and of the proper caliber.

The nerve graftings may be divided into three groups, as illustrated in Figure 2. Technically the operation is easiest in cases where the whole graft is situated within the facial canal and is most difficult if the peripheral ramifications have been injured rather than the stem of the nerve. The prognosis of the repair is closely related to the technical difficulties encountered.

3. *Nerve Suture.*—In very few cases is it possible to perform a direct intratemporal suture of the nerve. In Bunnell's "rerouting" method, the course of the facial nerve may be shortened by lifting it out of the Fallopian canal and suturing the stumps directly across the promontory (Fig. 2*D*).

Most authors, among them Cawthorne and Sullivan, prefer a graft to a suture after "rerouting" the nerve, because it leaves the nerve in the Fallopian canal with an undisturbed blood supply. Even if the results which I have obtained by following Bunnell's intentions have been satisfactory, the results of nerve grafting are so good that I, too, prefer this method.

Bunnell is, of course, quite right that after direct suture the neurofibrils will have to pass only one obstruction. But from practice we know that if only the coaptation is good excellent results can be expected from nerve grafting. It should be remembered that if one has to sacrifice important structures (the middle ear)—which could be avoided by using a graft—in order to perform a rerouting this operation is, of course, strongly contraindicated.

The choice, when there is one, between nerve grafting and nerve suture is not so simple as generally believed, and Lathrop

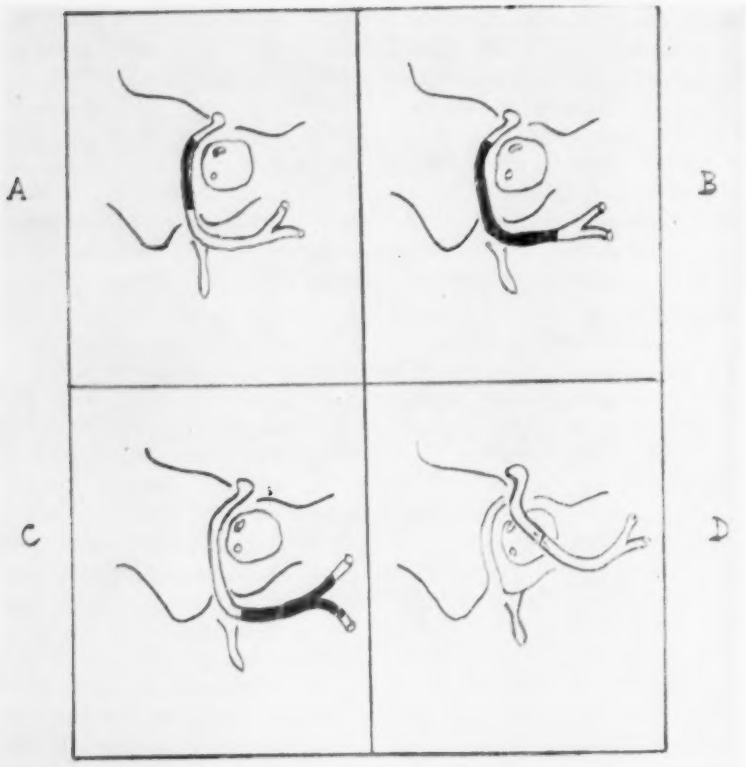



Fig. 2.—*A*, intratemporal nerve grafting according to Ballance and Duel. *B*, intra- + extratemporal nerve grafting. *C*, extratemporal nerve grafting. *D*, nerve suture after "rerouting" of the nerve according to Bunnell.

may be perfectly right when he says that Bunnell's rerouting method has not received the attention it warrants. My own experience with Bunnell's operation has been very satisfactory.

The after-treatment is of the greatest importance. The muscles must be supported while the nerve is recovering. Various hooks and splints have been recommended, but I quite agree with Sullivan that any fascia lata hook-up operation should be avoided during the stage of recovery. Massage and galvanism are of the greatest importance, and as soon as reinnervation starts active movements before a mirror should be instituted.

Two conditions must be fulfilled if a good result is to be expected:

1. No degeneration of the muscles must have taken place, as shown by a strong response to the galvanic current. By proper treatment (galvanization, massage, and various measures for supporting the relaxed muscles) these may be kept fully active for years, and thus repair may be justified at a late date.

2. The site of the lesion must be surgically accessible, which in practice means that the lesion is located distal to the geniculate ganglion. Valuable information as to the site of injury can be obtained by modern radiography, especially by the technique of Chaussé in Schüller's projection, by tomography of the temporal bone (Botman and Jongkees), and, in early cases, by applying Tschiassny's "topognosis."

The traumatic facial palsies may be divided into three main groups: I. Postoperative facial palsies. The majority of these

cases are due to mishap in otological surgery; others may result from operations on the neck and cheek (e. g., removal of parotid tumors), and some may follow extirpation of tumors of the posterior fossa (acoustic neurinomas). II. Palsies due to fractures of the temporal bone. III. Palsies resulting from lacerations of the face.

From a prognostic point of view the following points are common to all three groups: 1. If the palsy arises in direct conjunction with surgery or accident, the continuity of the nerve may be, and generally is, severed. The prognosis of reinnervation is then very bad if the nerve is not repaired. 2. If the palsy arises after an interval of freedom, the continuity of the nerve is not severed and the prognosis is on the whole fairly good.

To understand the reasons for the following indications, it should be remembered that the facial nerve within the temporal bone is enclosed in a rigid bony canal without any chance to expand or, in many cases, to escape injury. Collier in her papers rightly always stresses that

> the fundamental distinction between degenerative and nondegenerative lesions must be recognized. Conduction of the nerve impulse may be temporarily impeded by a transient block which affects the myelin sheath without leading to degeneration of the axons. Degenerative lesions are of two kinds depending on whether or not the supporting structures of the nerve—the Schwann cells and the neurolemma tubes—remain intact. Interruption of the axons with preservation of the supporting elements may be followed by spontaneous, though not perfect, recovery, as the regenerating axons can grow along the old paths, but if there is complete separation of the nerve elements spontaneous recovery is prevented. This variety of total nerve injury does not necessarily require macroscopic section of the nerve in its continuity. Excessive intraneural fibrosis can also destroy the Schwann cells and interrupt the neurolemma tubes. There is no clinical difference between these types of nerve paralysis, nor does electrical testing or electromyography reveal any difference.
>
> The rigid fallopian canal introduces a factor in the pathology of injury to the facial nerve which is not necessarily present in the nerves of the limbs. Nondegenerative lesions due to pressure on the nerve produce their effects by occlusion of the blood vessels. This ischemia affects primarily the myelin sheath and spares the axis cylinders and Schwann cells. If, however, the pressure is continued, vascular stasis occurs in neighboring segments leading to edema above and below the site of compression. The process of repair in the compressed area is impeded, and finally complete degenerative lesions involving the axis cylinder and the Schwann cells may be produced. It is necessary, therefore, to consider the changes in the facial nerve from the original compression, however produced, and those changes which may be brought about by persistent compression. At the present time we have no means of knowing whether ischemic block is likely to persist and go on to degeneration.

Concerning Bell's ischemic facial palsy, Collier has written as follows:

> The problem case is the one where the original injury has produced an interference with conduction which of itself is temporary and recoverable, a condition that is sometimes called neuropraxia, but where persistence of the compression in the rigid canal can produce changes which may lead to degeneration.

I profoundly agree with Collier. Further, what is written here with reference to Bell's palsy is equally applicable to situations which may be encountered in dealing with traumatic facial palsies. I should like to stress once more that at the present time we have no means of knowing whether ischemic block is likely to persist and go on to degeneration. That is why we sometimes are forced to do an explorative decompression of the facial nerve to prevent pressure on the nerve from leading to degeneration of the axons.

## I. Postoperative Facial Palsies

Any operation along the peripheral path of the facial nerve incurs the danger of injuring the nerve; such operations may be classified under three headings.

1. *Palsies Due to Removal of an Acoustic Neurinoma.*—In removing a neurinoma of the acoustic nerve it may be impossible to avoid a lesion of the facial nerve.

The site of injury is located intracranially which site is generally considered inaccessible for performance of nerve grafting. In most of these cases an anastomosis has been performed between the proximal end of another cranial nerve (accessorius, hy-

poglossus) and the peripheral stump of the facial nerve. As already stated, mimicry will never return after such a procedure.

It should, however, be mentioned that in a case of this type Dott successfully inserted a nerve graft, and that in a case of labyrinthine necrosis Tickle sutured the proximal end of a 16 mm. nerve graft to the proximal end of the facial nerve where it pierces the dura, and the distal end to the peripheral stump of the facial nerve within the Fallopian canal.

Indications: I feel then that our endeavors in palsies occurring in conjunction with the removal of neurinomas of the acoustic nerve should be directed toward repairing the nerve at the site of injury, and that we should not be content with the second-best solution, i. e., an anastomosis. Direct repair has, as just stated, been successfully performed, and I think that further work on this problem is imperative.

*2. Palsies Due to Mishap in Otological Surgery.*—The palsy most commonly arises in conjunction with a simple or radical mastoidectomy, a labyrinthectomy, or, more rarely, a fenestration operation. The indications for surgical intervention largely depend upon the time of onset.

Indications: (*a*) Palsies arising in direct conjunction with operation. In such cases, severance of the continuity of the nerve must seriously be suspected. Opinions, however, differ as to the course which should be taken. Collier is in favor of expectant treatment:

> Since a nerve graft may be necessary in these cases it may be urged that operation should be undertaken at once. There are, however, two good reasons for delay. In the first place infection in the neighbourhood of a graft or suture leads to fibrosis, both intra- and extraneural, which prevents the regenerating axons reaching the peripheral stump. Second, the outgrowth of Schwann cells from the peripheral stump towards the center, which plays the part of a bridge by which the regenerating axons reach the periphery, is most vigorous between 15 to 25 days after division. Delay, therefore, for about three weeks will favor satisfactory regeneration.

Bloch et al. agree with Collier's point of view. Collier may be perfectly right; however, my experience has told me that with local and general administration of antibiotics an existing infection is no contraindication to immediate repair of the nerve. Many of the otological injuries occur after a radical mastoidectomy performed in a case of chronic otitis, and, as pointed out by Sullivan, we are dealing here with a low-grade infection.

I have always maintained that if a palsy arises in direct conjunction with the operation the assumed site of the lesion should be inspected without delay. There is one way and only one way to learn what has caused the palsy and what remains to be done, and that is immediate inspection. This is the only sensible procedure to follow. Electromyography may at a later date reveal that something interferes with conduction but not the nature of the injury, and this may be of such a nature that the proper treatment should be instituted immediately. Here I am in agreement with Ballance and Duel, Cawthorne, and Sullivan. From experience I know how much easier it is to find the nerve stumps immediately after injury than weeks, months, or even years afterward, when they have to be separated from granulations and scar tissue. As previously mentioned, infection in the cavity is no contraindication to immediate repair.

(*b*) Palsies occurring after an interval of freedom. Here we know that the continuity of the nerve is unbroken and that the palsy may be due to pressure of a tamponade, to edema, or to hemorrhage in the facial canal. The prognosis is on the whole fairly good. The palsy may, however, also be due to progressive osteitis, necessitating a revision.

Because severance of the nerve in cases of delayed palsies can be excluded, electromyography may be an adjunct, which, however, should not be relied upon more than other methods of examination.

Consequently, if it is practicable to examine the patient electromyographically, I should not hesitate to do a decompression as soon as severe interference with conduction is demonstrated. If it is known for

certain that the nerve was severed, the repair may be postponed, even though I think that postponement is incorrect, as just mentioned. But the palsy may be due to an edema or a hemorrhage, which, as previously outlined, has produced an interference with conduction, which of itself is temporary and recoverable but may lead to degeneration, where compression in the rigid canal can persist. Therefore the compression should be relieved as soon as possible.

If no electromyograph is available, and if no signs of returning mobility are noted, decompression should not be postponed for more than two months at the utmost. From experience we have learned that in the favorable cases which recovered spontaneously and completely the first signs of spontaneous movements were noted before two months had elapsed, and generally within two to three weeks, regardless of whether the palsy was due to a middle-ear infection (Kettel), to a fracture of the skull (Rowbotham), or we were dealing with Bell's ischemic palsy (Arnvig).

Summarizing, it may be said that if a complete palsy arises in direct conjunction with the operation the supposed site of the lesion should be inspected without delay. If the palsy appears after an interval of freedom, a decompression should be considered as soon as severe interference with conduction is shown electromyographically or, if the apparatus is not available, after two months of observation at the utmost.

*Observations and Results.*—Only cases in which a complete peripheral facial palsy appeared in direct conjunction with operation are included in the following pages. The material comprises 97 patients.

A simple glance at the Table shows the justification of immediate exploration of the facial nerve in cases in which a complete palsy arises in direct conjunction with operation. In about two-thirds of the cases the continuity of the nerve proved to be severed. "Watchful waiting to determine whether or not the facial nerve has been injured permanently courts disaster, and

*Cases in Which Complete Facial Palsy Appeared in Direct Conjunction with Operation on the Temporal Bone*

| Operation | Continuity of Nerve | | Total No. |
|---|---|---|---|
| | Severed | Preserved | |
| Simple mastoidectomy | 21 | 6 | 27 |
| Radical mastoidectomy | 36 | 25 | 61 |
| Labyrinthectomy | 4 | 1 | 5 |
| Fenestration | 3 | 1 | 4 |
| Decompression of the facial nerve, seq. | 1 | 0 | 1 |
| Total | 65 | 33 | 98 |

may condemn the patient to a life of embarrassing deformity," says Maxwell, and I agree wholeheartedly with his opinion.

Simple Mastoidectomies: In these operations the facial nerve is injured (*a*) by creation of an artificial antrum below and distal to the horizontal canal and (*b*) in removal of the retrofacial group of cells in an extensively pneumatized bone (Sullivan).

According to the condition of the nerve the cases may be divided into two groups.

A. Continuity of nerve preserved. This group comprises six patients. Unless otherwise stated, the palsy was complete when decompression was undertaken. Cases 1, 4, 5, and 6 were referred to and operated on in Frederiksborg County Hospital here. The remainder I operated on in that hospital in which the mastoidectomy had been performed. In all these cases decompression was done.

Case 1.—Boy, 2 years of age. Palsy was noted immediately after operation. The wound was reopened and the nerve inspected. It had been denuded in the vertical segment and partly decompressed. It looked entirely normal. Complete recovery followed.

Case 2.—Girl, 10 years of age. In every respect like Case 1.

Comment: These patients would have recovered spontaneously, but a severance of the nerve could not be excluded, and therefore inspection was absolutely indicated.

Case 3.—Girl, 3 years of age. Exploration one week after the mastoidectomy. There was considerable osteitis in the posterior osseous wall and the facial canal, and the nerve was highly edematous. On reexamination one year later she looked normal and was able to screw up her eye, but the smile was somewhat limited and she was unable to wrinkle her forehead.

Comment: Even if she did not recover completely, the value of decompression can hardly be questioned, as an area of pronounced osteitis around the nerve, which might have destroyed the nerve, was excochleated.

Case 4.—Girl, 4 years of age. Referred four months after the mastoidectomy. The nerve had been denuded and superficially injured by a curette. A neurolysis was performed. On reexamination five years later she looked normal with the face at rest and was able to screw up her eye, but the smile was limited.

Comment: This patient might have recovered to the same degree spontaneously, but in this case also a severance of the nerve could not be excluded.

The following two patients differ from those mentioned above, as they were referred years after the mastoidectomy, with partial mobility of the injured side of the face.

Case 5.—Woman, 32 years of age. Referred to Frederiksborg County Hospital five years after the mastoidectomy. The mobility of the face on the injured side was rather poor, and a slight contracture was noted. With the greatest reluctance I explored the nerve, which was found denuded in the vertical segment, looking atrophic. I have not seen the patient since, but one year later she reported that "the difference in mobility between the two sides was not nearly as pronounced as it had been before decompression."

Comments: She may be right in her evaluation of the result, but as I have not examined her personally I shall only state that probably no improvement followed. But we may wonder what caused the atrophy and whether any damage could have been repaired by immediate exploration.

Case 6.—Man, 19 years of age. Referred to F. C. H. eight years after the mastoidectomy. He was unable to wrinkle his forehead; he could close his eye but not screw it up, and he was unable to show his teeth. The mobility was very poor, but faradic current evoked violent contractions of all the muscles, indicating that no atrophy had taken place. On decompression the nerve was found denuded in the vertical segment, surrounded by scar tissue, and a neurolysis was performed. On reexamination four years later he looked normal with the face at rest and he could screw up his eye, smile to some degree, and show his teeth.

Comments: This shows that even years after injury a neurolysis may improve the condition.

One and only one thing can be deducted from these operations: Immediate inspection of the nerve should be done if a complete palsy occurs in direct conjunction with operation. It may be that the nerve is only denuded and not damaged, or it may have been slightly or more severely injured without being completely severed. Further, perhaps the mastoidectomy has been incomplete, leaving behind an osteitis of the facial canal. But above all, the possibility of a severance of the nerve cannot be excluded, and this alone justifies immediate exploration.

If the patient is seen years after operation a neurolysis may improve the condition (as in Case 6), but at this time decompression should be done only after serious consideration. It may be helpful, but it may also be without any effect at all, and it should also be remembered that even in experienced hands damage may be done. I once performed a decompression on a patient suffering from Bell's ischemic palsy who had partially recovered, and her condition was impaired after the operation. The second case of this nature which I treated will be described later.

B. Continuity of nerve destroyed. To this group belong 20 patients. The interval between mastoidectomy and repair varied widely.

In all cases the nerve had been injured in the vertical segment. In two cases continuity was partly destroyed and in 18 it was completely destroyed.

Nerve grafting was performed in 15 cases and neurolysis in 1 case. After refreshening of the nerve stumps, a graft varying in length from 6 to 35 mm. was inserted. In four cases repair was done within a week after injury; in seven cases 3 to 5 months later, and in two cases 18 and 22 months after the mastoidectomy. In all these cases grafting was performed with success. In the last two cases repair was undertaken 4

and 30 years, respectively, after injury, but without success, as will be described later. In some cases the gap was only short and the whole graft could be placed strictly intratemporally. In others the destruction was so extensive that the peripheral end of the graft had to be sutured to the distal stump of the facial nerve outside the stylomastoid foramen.

In the first two cases the continuity of the nerve was only partly severed, and they need comment.

CASE 7.—Boy, 10 years of age with complete facial palsy. Exploration of the nerve was done three and one-half months after the accident. The lateral half of the nerve in the vertical segment had been cut, and the nerve was surrounded by scar tissue. A neurolysis was performed. On reexamination eight months later he looked normal, was able to screw up his eye, but his smile was somewhat limited, and he was unable to wrinkle his forehead.

Comment: If the nerve had been explored immediately, a lateral graft should have been inserted in the gap, and a better result might have been expected.

CASE 8.—Boy, 5 years of age. Repair was done five months after injury. The nerve had been denuded in two places: 1. It was denuded 5 mm. proximal to the stylomastoid foramen, where three-fourths of the fibers were cut, with formation of neuromas on the stumps; over a distance of 10 mm. there was only a tiny strand of nerve fibers. 2. Just distal to the lateral semicircular canal it was also laid bare, lying imbedded in scar tissue. The nerve was decompressed as far as the geniculate ganglion. The whole nerve was flaccid, hanging down over the promontory like a loose string. As the continuity was not completely broken, what remained of the nerve was conserved, and additionally a 32 mm. end-to-side graft was inserted from the geniculate ganglion down to just outside the stylomastoid foramen. On reexamination three years later the patient looked normal with the face at rest, but could move his mouth only a little and not close his eye completely.

Comment: Two errors were committed in this case. First, the nerve should have been inspected immediately, not five months later. A proper repair could then have been done. Second, when I was called in I ought to have cut the whole atrophic part of the nerve and inserted a graft. Both of these procedures, especially the first, would, no doubt, have given a better result. As Collier has stressed, "total nerve injury does not necessarily require macroscopic section of the nerve in its continuity. Excessive intraneural fibrosis can also destroy the Schwann cells and interrupt the neurolemma tubes."

If, moreover, the nerve at the site of injury looks atrophic, there is every reason to believe that this part of the nerve has been transformed into connective tissue, which should be removed and replaced by a graft. It is impossible to say whether the mobility present three years later was due to the preserved part of the nerve or to the graft.

Decompression was absolutely indicated in the two cases described above, as a severance of the nerve could not be excluded, but it is open to discussion whether the patients recovered on account of the nerve grafting or whether the mobility is due to preserved neurofibrils.

This reservation does not apply to the next 13 cases, as the continuity of the nerve was completely destroyed in all of them. From previous investigations we know that in such cases a lifelong complete palsy results (Kettel, Riskær).

The results of nerve grafting in 15 cases may be summed up as follows:

1. A clinically satisfactory result was obtained in 13 cases: All the patients looked normal with the face at rest. Twelve could screw up the eye and smile, both movements at approximately 75% of the function on the normal side. One patient could only smile, not close the eye. None of the patients could wrinkle the forehead.

2. Some innervation, but clinically quite insufficient, was noted in one case.

Woman, 36 years of age. Thirty years ago (!) a complete facial palsy followed a simple mastoidectomy. She still had a complete facial palsy and a large retroauricular fistula. Thanks to electrical treatment over the years, there was still some contractibility of the muscles. There was nothing to lose, and perhaps a little to gain, by operation. In closing the fistula the nerve was

inspected simultaneously. It had been cut at the second bend. Not till just distal to the geniculate ganglion and just proximal to the pes anserinus did the nerve look comparatively normal. A 35 mm. graft was inserted, and in two years some reinnervation took place, but it was quite insufficient, and a plastic operation was performed.

Comment: No wonder that the "result" was of practically no value; the conditions for repair were too bad for anything to be expected.

3. No reinnervation (one case).

A man 28 years of age was referred four years after injury. A tiny atrophic strand connected the stumps of the facial nerve from just distal to the second bend of the nerve to a point just proximal to the stylomastoid foramen. It was preserved; the sheath was split, and an end-to-side graft was inserted. No improvement resulted.

Comment: In this case I should have resected the atrophic part of the nerve and inserted an end-to-end graft. My decision to preserve the string was a poor one.

Nerve suture. In the majority of cases it is necessary to perform a radical mastoidectomy in order to suture the nerve after "rerouting" it. Therefore, in repairing an injury to the vertical segment of the nerve, Bunnell's method should be considered only if the middle ear has already been damaged or if it must be sacrificed in order to get access to the sound proximal end of the facial nerve.

These conditions were fulfilled in four cases referred to me for repair.

In three cases in which suture was done one week, three months, and eight months, respectively, after injury a clinically good result was obtained. The palsy was not noted with the face at rest; two could smile and screw up the eye, and one could only smile.

In the last case repair failed:

Girl, 10 years of age. Eight years previously a complete palsy had resulted from a simple mastoidectomy for a chronic tuberculous mastoiditis. The palsy was still complete, but as there was a large secreting retroauricular fistula, which had to be closed, and a fair response to the galvanic current, a revision was made. The middle ear was completely destroyed, and the nerve had been cut. The refreshened ends were approximated across the promontory, but for technical reasons it was impossible to pass a suture through the very short proximal end, which remained distal to the geniculate ganglion.

Comment: Whether the failure was due to poor coaptation of the nerve ends or to the tuberculous patient's general state of health combined with partial degeneration of the muscles of the face, it is impossible to say.

Summarizing, it may be said that in 17 of 19 patients who suffered from a complete palsy in conjunction with a simple mastoidectomy the conditions for repair were good, meaning that no atrophy of the muscles had taken place and that the nerve stumps could be isolated. In 16 of these 18 patients a clinically good result was obtained by nerve grafting or suture. In one repair failed because the wrong operation was done. In the remaining case a fair result followed neurolysis. Without repair of the nerve all the patients would undoubtedly have had a lifelong complete palsy.

In two cases the conditions for repair were too bad for a satisfactory result to have been expected.

Radical Mastoidectomies: In 61 patients a complete peripheral facial palsy developed in direct conjunction with the operation. The cases may be divided into two groups according to the condition of the nerve.

A. Continuity of nerve unbroken. This group comprises 25 patients, in 20 of whom a fistula on the facial canal was observed on decompression.

(*a*) Fistula on the facial canal with nerve of normal appearance.

To this group belong six patients, in five of whom exploration was made within two days of the radical mastoidectomy. The remaining case was explored four days after the operation. The fistula was widened (partial decompression), but no osteitis was seen in the wall of the facial canal and the nerve looked normal.

On reexamination, all the patients looked normal with the face at rest. Four had recovered completely; one had only minor and one a more pronounced impairment of function of all three branches.

(*b*) Fistula on the facial canal, nerve covered by granulations.

This observation was made in five cases, but on partial decompression the wall of the facial canal appeared normal. Exploration was performed 0, 0, 21, 28, and 28 days after the radical mastoidectomy. Three had recovered completely, and one patient died years later of a liver disease before reexamination. One patient was left with significant impairment. He looked normal with the face at rest and was able to screw up his eyes, but when he smiled a difference could be noted between the two sides.

*(c)* Fistula on the facial canal, nerve heavily edematous.

This was observed only twice. In one case decompression was done immediately and the nerve sheath was split. This patient recovered completely. In the other, decompression was not undertaken until four months later. On reexamination the patient was unable to wrinkle his forehead and close his eye, and his smile was poor.

*(d)* Fistula on the facial canal, nerve covered with granulations and showing progressive osteitis in the wall of the facial canal.

To this group belong seven patients. In five cases exploration was done one week after the radical mastoidectomy, in one three weeks later, and in the last case two months later. On reexamination, all of them looked normal with the face at rest. One patient had recovered completely, five had only minor defects, and the last had a rather pronounced impairment of function.

*(e)* Intraneural hematoma of the facial nerve.

In these two cases the palsy probably did not appear until a few hours after the operation, but this is hard to say with certainty in a patient recovering from an anesthetic. Therefore they are included in this series.

In the first case the patient was observed for three months, but as the palsy was still complete, decompression was done. Just distal to the lateral semicircular canal the nerve sheath looked thickened (organized hematoma?) and was therefore split. The first signs of returning movement were not noted until seven months later, and the patient had residual impairment, even though he looked normal with the face at rest. In the second case the reaction of degeneration was almost complete a week after the radical mastoidectomy, and the nerve was then uncovered. An intraneural hematoma was found just distal to the lateral semicircular canal, and on splitting of the sheath the nerve bulged outward. Movements started 11 days later, and 4½ months after decompression the patient had recovered completely.

*(f)* Fracture of the facial ridge.

In three patients a fracture of the apex of the facial ridge followed chiseling down of the posterior osseous wall in a radical mastoidectomy.

One patient was referred for repair eight months later with a complete palsy. A sequestrum was compressing the pyramidal segment of the nerve; it was removed, and on splitting of the sheath the nerve bulged greatly. Movements started three weeks later. After two years she looked normal, could screw up her eye and smile to a limited degree. The forehead, however, was immobile on the injured side.

One patient was referred four years after injury. There were hardly any movements, even though the palsy was not noted with the face at rest. A sequestrum was found compressing the pyramidal segment of the nerve, which was extremely atrophic and imbedded in granulation tissue.

Hardly any improvement followed.

In the last case, operated on recently, several bony spicules were compressing the nerve just at the second bend and the nerve was heavily contused.

Summarizing the results of decompression in cases of complete palsy in direct conjunction with a radical mastoidectomy, but without destruction of the continuity of the nerve, they may be divided into three groups.

1. Manipulations on the fistulous facial canal. Nineteen of twenty patients have been reexamined; one died of a liver disease. All the patients looked normal with the face at rest, nine patients had recovered completely, and six had minor and four more pronounced impairment of function. Some of the patients would without doubt have recovered to the same degree spontaneously, especially those where the nerve looked macroscopically normal.

In seven cases a progressive osteitis had crept via the fistula into the facial canal and partly destroyed it. After removal of the osteitis of the wall by decompression of the nerve until it looked normal without any exterior signs of infection, one patient recovered completely, five had minor defects, and in one there was a rather pronounced impairment of function. Personally I think that these results have been obtained because of decompression and that they would have been far worse if the area of osteitis had not

been removed and the nerve had become involved.

2. Hemorrhage in the facial nerve. In one patient decompression was postponed for three months, and he did not recover completely. In another, decompression was done one week after the radical mastoidectomy, and she made a complete recovery, due, I believe, to the early intervention.

3. Fracture of the facial ridge. The sooner fragments compressing the nerve are removed, the better. In one case, operated upon eight months after injury, a satisfactory result was obtained, whereas in a second case an attempt at repair four years later failed because the nerve was atrophic. The third patient was operated on only recently.

In many of these cases it is open to discussion whether recovery took place on account of or in spite of decompression. But the important lesson is that only by inspection can the nature of the lesion be established. If the nerve is severed, the earlier repair is undertaken the better, but most important is the possibility that the palsy may be due to compression by edema, bleeding, infections, or fragments of bone. If this pressure is relieved, degeneration may be avoided; if not, a condition which in itself is reversible may lead to degeneration. This fact alone justifies inspection of the nerve. Explorative operations are imperative in many fields of surgery, and surgery of the facial nerve is certainly not an exception. Expectant treatment means taking chances.

B. Continuity of the nerve destroyed. This group comprises 36 patients. The nerve may be injured in planing down the posterior osseous wall, in removal of granulation tissue from the middle ear when a fistula on the facial canal is present, and in failure to identify the horizontal segment of the facial canal.

Whereas it may be difficult to decide for or against decompression in cases where the continuity of the nerve is unbroken, this becomes very easy when it is severed, as reinnervation never occurs when the nerve is cut.

In 32 cases a nerve graft was inserted, and in 3 cases a nerve suture was performed. In 13 patients repair was undertaken within a week after injury, in 14 after this time but within 2 months, in 5 between 2 and 12 months, and in the remaining 4 after this time.

The length of the graft varied from 4 to 50 mm.; in many cases the peripheral end had to be sutured to the facial nerve in the soft tissues outside the stylomastoid foramen.

The minimum requirements for the result to be classified as good are that the palsy must not be noted when the face is at rest and that the patient can smile and laugh to a certain degree. It should be borne in mind that not even the most successful operation will be able to restore mimicry completely, but, remembering that it is unanimously agreed that without repair all these patients would be doomed to a lifelong embarrassing deformity, the results of nerve grafting and suture are most gratifying.

Six patients have been operated upon recently. In 31 cases the treatment is finished, and all the patients have been reexamined. The results are as follows: In 27 cases (24 graftings, 3 sutures), a clinically satisfactory result was obtained. All of them looked normal with the face at rest; 25 could smile and screw up the eye, and 6 among the latter could also wrinkle the forehead a little. The last two patients (graftings) could only smile.

In one patient some movement returned, but it was clinically quite insufficient. In three cases the attempt at repair failed completely. One of the failures may be due to the fact that partial degeneration of the muscles had taken place at the time of repair, but I cannot offer an explanation for the failure in the other two cases. The coaptation may have been poor, as the operations were done before I started using magnification.

Labyrinthectomies: In five cases a complete palsy occurred in direct conjunction with labyrinthectomy. In two of these the continuity of the nerve was completely severed; in two it was partially severed, while in one it was unbroken.

A. Continuity of nerve unbroken.

Man, 20 years of age. In this patient exploration had to be postponed for three weeks on account of leptomeningitis. The facial canal was fractured near the apex of the facial ridge, and a fragment was compressing the discolored and contused pyramidal part of the nerve. Two years later the palsy could not be noted with the face at rest, and he could screw up his eye and smile, but not to the full extent.

Comment: If the impinging piece of bone had been removed immediately, the result might have been better.

B. Continuity of nerve destroyed.

In two cases a 10 mm. graft was inserted. In one patient, a youth 18 years old, the result was good, as the palsy could not be noted with the face at rest, and the patient could screw up his eye and smile to some extent. In the other case, a man 31 years old, some reinnervation took place, but it was clinically quite insufficient. I am unable to explain this failure. I expected the patient to make a good recovery.

The other two cases deserve more detailed comment.

Woman, 24 years of age, two years after labyrinthectomy was referred with the corner of the mouth practically immobile, unable to close her eye or wrinkle her forehead.

From 5 mm. distal to the geniculate ganglion to 5 mm. proximal to the stylomastoid foramen, the nerve was transformed into an atrophic strand. This was preserved, and the nerve was uncovered until its caliber was normal. A 25 mm. graft was inserted after the part of the nerve outside the connecting strand had been refreshed. Movements started four months later, and a year later she looked normal, and could smile and screw up her eye.

Comment: Even though the nerve had not been completely severed, the tiny strand between the sound proximal and distal parts of the nerve had not been able to restore innervation of any practical value. It confirms, however, what I have stated earlier: "If only the slightest connection remains between the proximal and distal parts of the nerve, this suffices to keep the oral muscles at a degree of tone that enables the angle of the mouth on the defective side to be at about the same level as the angle on the unaffected side." I have been able to confirm this statement on several occasions, where my endeavors to improve the condition were fortunately successful. Lately McGillicuddy has arrived at the same conclusion in a case of 20 years' duration.

The remaining case should be reported in detail, because of its very unfortunate outcome, which illustrates that much damage may be done even by one who is comparatively well trained in these operations.

A man 24 years of age was admitted to the hospital on Oct. 8, 1945. In 1927 he had been admitted to another clinic with chronic otitis media and preoperative facial palsy. First a radical mastoidectomy was performed, and one month later a labyrinthectomy. Two months later a revision was done. On his admission in 1945, the palsy was hardly noted when the face was at rest, but he was unable to close his eye or wrinkle his forehead and could move the corner of his mouth only a very little. The reaction to the galvanic current, however, was strong. In the radical cavity, pus and granulations were observed.

Because of his inability to move the corresponding side of the face sufficiently and the strong response to the galvanic current, an attempt was made to repair the nerve. The cavity was cleaned of granulations, and the facial nerve was isolated at the stylomastoid foramen. As far as the bend of the lateral (removed) semicircular canal it looked normal, but the tympanic (horizontal) part was extremely thin and could be followed only to a point near the geniculate ganglion; there it disappeared. In spite of great care, I must have damaged this tiny strand, and as all anatomical landmarks were missing on account of the previous operation, I was unable to locate the proximal end of the nerve. A graft was placed proximally at the supposed site of the geniculate ganglion and connected with the peripheral stump of the facial nerve. Complete palsy followed, and movements did not return. I have learned that later an anastomosis operation proved a failure, and that plastic operations were performed.

Comment.—In this case severe harm was done to the patient. On arrival, his face looked normal, even if he could move the afflicted side of the face only a very little.

My efforts to improve the condition resulted not only in complete failure but in a pronounced impairment.

Summarizing, a clinically satisfactory result was obtained in three cases, once by removal of a fractured piece of bone compressing the nerve and twice by nerve grafting. In one case of nerve grafting no reinnervation took place, and in the last case severe damage was done, as described above.

Fenestrations: In fenestrations the facial nerve is most commonly injured by the burr in creating the fistula. Four cases of complete palsy were referred for repair. In one case the continuity of the nerve had been preserved, while in three it was destroyed.

A. Continuity of nerve unbroken.

Man, 30 years of age. Fourteen months previously, a fenestration had been done elsewhere. The labyrinth was destroyed, and immediate facial palsy followed. On admission the palsy was still complete, and an exploration was done. The horizontal part of the nerve had been denuded and was swollen but not severed.

Comment: By the inspection it was ascertained that the continuity was preserved. Two years later he had improved considerably, and probably spontaneously.

B. Continuity of nerve destroyed.

In three cases a complete palsy occurred in direct conjunction with operations performed elsewhere. They were referred for repair 2, 3, and 10 months, respectively, after the accident.

In two cases, part of the horizontal segment of the nerve had been removed. In the third case even most of the vertical part was missing. Grafts ranging from 8 to 20 mm. were inserted. In two of the cases a clinically satisfactory result was obtained.

I did not see the last patient until six years later in another hospital, where through the courtesy of the physician-surgeon in charge I had the opportunity to read the case record and learned that four years after nerve grafting there was a good function of the mouth, although the patient was unable to close his eye and wrinkle his forehead. Shortly afterward a revision had been performed because of granulations in the cavity, and in immediate conjunction with this operation complete palsy returned. Three years later the palsy was still complete. There can hardly be any doubt that at the revision operation the graft was unfortunately removed.

Summarizing, four cases of complete facial palsy occurring in direct conjunction with a fenestration operation were referred for repair. Decompression was done in one case and nerve grafting in three. A clinically satisfactory result was obtained in three cases; in the fourth good reinnervation had taken place but was destroyed again in an unfortunate later intervention.

Sequelae to decompression of the facial nerve: One patient suffering from Bell's ischemic palsy, "rheumatic facial palsy," had been subjected to decompression elsewhere and the nerve had been cut.

A man, 21 years of age, for one year had been suffering from Bell's palsy, which was still practically complete. Thirteen months after the onset, decompression of the facial nerve was performed elsewhere, but with no effect. He was referred two years after onset of the palsy, or one year after decompression had been done.

He had complete palsy, partial atrophy of the muscles, no faradic response, and diminished response to the galvanic current.

The conditions for repair were poor, but there was nothing to lose. The nerve had been cut in the middle of the vertical segment. The stumps were atrophic and had to be refreshened. A graft 30 mm. long was inserted. Six years later some reinnervation had taken place, but it was clinically quite insufficient.

Comment: Operation on the facial nerve is not a routine procedure of an otologic department, and should be done only by otologists especially trained for this purpose. The first decompression should not have been postponed for a year. When the repair was undertaken, conditions were so unfavorable that very little could be expected. The patient had, of course, been warned of this in advance.

3. *Palsies Due to Extratemporal Operations.*—Any operation on the face along the course of the nerve may produce a facial palsy, but the majority of facial palsies are

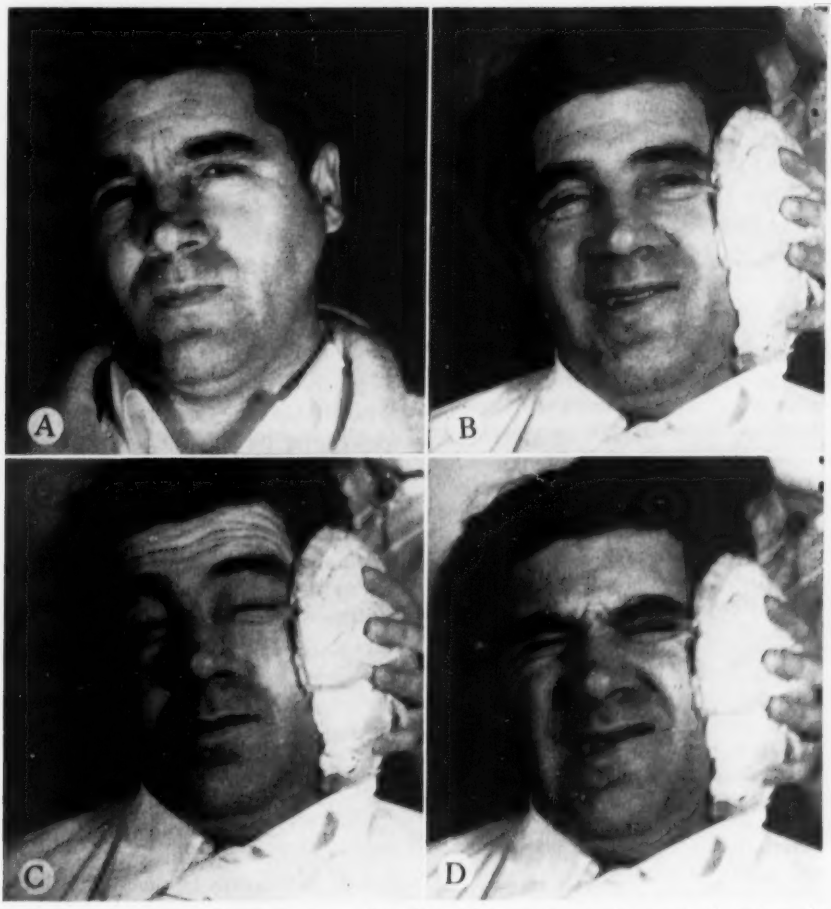



Fig. 3.—Large mixed tumor of the deep lobe of the left parotid gland. *A*, before the operation. *B-D*, three days after removal of the tumor. Only the mandibular branch of the facial nerve is paralyzed.

due to removal of tumors of the parotid gland. Many of these palsies can be avoided by careful dissection, as the nerve does not penetrate the substance of the gland but is situated between the superficial and the deep lobe (Fig. 3).

Indications: Prevention is better than cure. Everything should be done to avoid injury to the facial nerve by isolating it from the start in removal of a parotid tumor. But if damage is done in spite of every precaution, the nerve should be repaired immediately. If exploration has, for one reason or another, been delayed, then it should be done as soon as possible. The longer repair is postponed, the more difficult is identification of the ramifications in scar tissue. Moreover, these will be transformed into connective tissue and thus be practically useless as pathways for neurofibrils traveling from the proximal end of a cut facial nerve along a graft into the denervated muscles.

To this group belong five cases. In four cases a complete facial palsy appeared in direct conjunction with extirpation of a tumor of the parotid gland, while in one case the palsy was due to incision of an abscess at the angle of the mandible.

Observations and Results: The first three of the four patients were referred to

F. C. H. with a complete peripheral palsy, one and one-half, two, and three years, respectively, after the tumor of the parotid gland had been removed elsewhere. The patients had been told in advance that the repair was to be regarded as an experiment, the outcome of which was impossible to predict. There was everything to gain and nothing to lose. Complete failure followed in all three cases.

Case 1.—Woman, 47 years of age. One and one-half years after the first operation the facial nerve was isolated just outside the stylomastoid foramen, where it had been partly cut. Nerve grafting had to be given up on account of severe scar formation in the cheek, which made identification of the ramifications of the nerve impossible.

Case 2.—Man, 47 years of age. Repair was undertaken three years after the first operation. The operative findings were exactly like those in case no. 1. A 100 mm. graft was united to the facial nerve just outside the stylomastoid foramen, and, as the peripheral ramifications of the facial nerve could not be identified in the masses of scar tissue, a tunnel was made subcutaneously and the graft planted directly in the muscles, in accordance with the technique of Cardwell.

Case 3.—Woman, 46 years of age. Two years after the first operation the facial nerve was isolated and found to be cut about 1 cm. proximal to the pes anserinus. Neurinomas on the nerve stumps were removed, and microscopy showed that the nerve had been transformed into dense scar tissue. A graft was inserted, but no improvement followed.

Case 4.—Girl, one year old, was referred two weeks after removal of the tumor. The nerve was isolated from the stylomastoid foramen past the division at the pes anserinus. It was surrounded by tumor masses, and one of the larger temporofacial ramifications had been cut and was sutured. Six months later mobility started to return, and on reexamination 14 months later she looked normal with the face at rest and she could wrinkle her forehead, screw up her eye, and smile to a limited degree.

In the last case the palsy was due to incision of an abscess at the left angle of the jaw.

Case 5.—Woman, 18 years of age. The incision was made when she was 3 weeks old. For many years she had been treated by galvanization, and, as reaction to this remained positive, an attempt to repair the nerve was made. The nerve had been cut, and neuromas were found at the stumps. These were removed, but on microscopy the adjacent part of the nerve was seen to be severely fibrosed. A 20 mm. graft was inserted, without any result, and plastic operations were later performed.

Thus my own attempts to repair facial palsies due to extratemporal operations by nerve grafting resulted in four complete failures, because the repair had been postponed for years. Either it was quite impossible to isolate the peripheral ramifications in scar tissue or they were themselves transformed into scar tissue, useless as a pathway for neurofibrils.

In only one case was a satisfactory result obtained, but here the repair was done three weeks after the initial operation. Only one of the major ramifications had been cut, and it was sutured successfully.

These very poor results should be an encouragement to repair the nerve immediately (Maxwell) rather than to adopt an attitude of masterly expectancy.

If repair fails, the only resort is to plastic operations.

## II. Palsies in Fractures of the Temporal Bone

*Diagnosis.* 1. Fracture: Fractures of the temporal bone involving the ear and the facial canal may be divided into two main groups: transverse and longitudinal.

The transverse fractures are primarily posterior fossa fractures which have resulted from terrific force applied through the condyles (Maxwell and Magielski). Cochlear and vestibular functions are lost, and, according to statistics, transverse fractures cause a facial paralysis in about 50% of the cases (Grove).

The longitudinal fractures are much commoner. According to Rowbotham, they are apt to extend from the vault to the base, starting in the temporal squama or the parietal bone and extending through the mastoid into the middle ear. There is bleeding from the ear, and the hearing may initially be impaired but the otic capsule is not destroyed. Facial paralysis occurs in 10%-18% (Grove).

To these two main groups, however, may be added a third group: a fracture restricted to the mastoid process itself (Ramadier and Caussé). This may result from various lesions (Lathrop, Myers).

But from the point of view of the immediate and delayed danger to life, and that of damage to cochlear and vestibular function, the longitudinal fracture has a far better prognosis than the transverse. Exactly the same applies to facial paralysis which may occur in conjunction with a skull fracture.

Roentgenograms, and especially tomograms, of the temporal bone may give valuable information, but in some cases it is impossible to see on the films a fracture line which is easily detected by operation.

There is, however, general agreement that facial paralysis associated with acute head trauma indicates temporal bone fracture in nearly every instance (Maxwell and Magielski).

2. Facial nerve: Facial palsies may be partial, but they are usually complete. They can be divided into two main groups according to the history of onset:

(*a*) Palsies developing in direct conjunction with the accident. These are, as stated by Grove, usually due to a lesion of the nerve by the line of fracture, either in the Fallopian canal or at the internal auditory meatus. Usually the nerve is injured distal to the geniculate ganglion (Rowbotham). They are usually complete, caused by traction, tearing, compression from extra- and intraneural accumulation of blood, or actual section of the nerve. They may also be due to a commotion of the nerve (Garcin).

The prognosis varies according to the character of the lesion, as will be discussed later.

(*b*) Palsies occurring after an interval of freedom. Here we know that the continuity of the nerve is preserved. These palsies generally are due to edema or bleeding within the Fallopian canal. The prognosis is generally fair, but lasting palsies may result on account of degeneration of the nerve caused by a pressure within the Fallopian canal which has not been relieved in time.

It should be remembered that it is not always easy to tell whether a facial palsy occurring in conjunction with a head trauma has developed immediately or after a short interval of freedom because extensive lesions often make examination difficult.

Whereas it is easy to see whether the paralysis is complete or partial, it is often impossible to be sure of the prognosis in complete palsies, as the mechanism varies. The palsies may be due to bleeding, edema, tearing, or even section of the nerve. Electromyography can show whether the conduction block is complete but cannot reveal the cause. Serial electromyograms may reveal whether the conductive power of the nerve is improving or not, but a period of waiting is not without risk. If the nerve is sectioned, it may be repaired later; but if the palsy is due to an intracanalicular bleeding or edema with pressure upon the nerve and its vessels, this may lead to irreversible degeneration, during the period of observation. This could, perhaps, have been avoided by decompression at an earlier date.

I quite agree with Maxwell and Magielski that electromyographic studies are valuable but have not proved to be infallible.

As for the faradic test, a positive response is a good prognostic sign, but not very much can be deduced from a negative one (Kettel, Maxwell and Magielski).

Regarding the site of lesion, roentgenograms, and especially tomograms, of the temporal bone may be valuable, but in many cases it is impossible to obtain concrete information in this way. Tschiassny's "topognosis" is, in my opinion, reliable only in early cases, and most of the patients are referred too late for this method to be used.

## Prognosis and Indications

### Review of the Literature

Up until now it has been impossible to agree upon the proper treatment of facial palsies in skull fracture. The attitude adopted by various otologists varies from

pure conservative treatment to unlimited surgical intervention.

I. *Advocates of Conservative Therapy.*—Turner is in favor of conservative therapy. He reported its use in 70 consecutive cases, in 36 of which the palsy was immediate; but a closer analysis shows that only 75% recovered completely. In 25% remnants of the palsy could be noted, varying from slight impairment of function to complete and lasting palsy.

In 34 cases the palsy appeared after an interval of freedom, and 32 of these patients recovered completely.

Coleman and Browder also advise conservative treatment in view of the large percentage of cases in which there is spontaneous recovery.

The same view is held by Kahn and Gurdjian, as, in their opinion, 90% of the cases recover completely or at least adequately.

II. *Advocates of Surgical Intervention.*—A few authors recommend operative treatment in the majority of cases; most surgeons, only in selected cases. Spurling states that the prognosis for complete recovery of immediate cranial nerve palsies due to skull fractures is poor.

Ersner recommends active intervention, as decompression of the Fallopian canal permits the escape of fluid, allowing edema of the nerve to subside, or makes possible repair of the nerve if the continuity is broken.

Farrior and Caldwell stated that "early exploration of any facial paralysis resulting from fracture of a temporal bone is the treatment of choice."

Behrman states that there is no reason to put in a special class those paralyses which occur in skull fractures. They ought to be dealt with as other traumatic palsies.

Cawthorne says that "skull fractures in which facial paralysis is accompanied by signs of damage to the ear, particularly if the middle ear is affected, should be considered as possible instances of fracture involving the middle ear cleft and the facial nerve as it runs along its inner wall." Recently Cawthorne has made an additional statement: "In a case of facial palsy, dislocation of the incus as evidenced by conductive deafness and in abnormal appearance of the tympanic membrane is a definite indication for exploration of the nerve trunk in the Fallopian canal." I completely agree with Cawthorne, and, moreover, I should like to add that a case in which a fracture line is seen in roentgenograms running from the temporoparietal area downward into the external auditory meatus should be considered as a possible instance of fracture of the facial canal, with damage to the nerve near the second bend.

Maxwell and Magielski state that, if after three months, or earlier, the palsy remains complete and there is complete reaction of degeneration, surgical exploration must be considered, provided the patient's general condition is good.

Rowbotham states that favorable cases show signs of improvement within two months and usually within two to three weeks. He concludes that although theoretically routine decompression should be done in all cases, particularly the delayed ones, early operative interference is contraindicated because of the high percentage of good spontaneous recoveries. However, he does advocate exploration of the nerve, regardless of electrical reactions, after three months in those patients who have shown no improvement.

Grove and Lang state that, even if the majority of the immediate palsies disappear spontaneously, surgery is absolutely indicated in a limited number of cases.

This survey shows that, even if conservative treatment should be adopted in the majority of cases, surgical intervention is imperative in selected cases.

I, too, have recommended this line of action, which I shall now discuss more thoroughly.

### Own Indications

I. *Immediate Palsies.*—It is my opinion that in cases of immediate and complete paralysis an exploration of the facial nerve

should be done as soon as the patient's general state of health permits it, once a definite diagnosis of a severe and accessible lesion of the nerve has been made.

By a severe lesion is to be understood complete conduction block with denervating potentials and fibrillation as indicated electromyographically. If an apparatus for electromyography is not available, I consider any palsy severe which shows no signs of spontaneous improvement within two months of observation, as the favorable cases show signs of improvement within this time limit, and usually within two to three weeks (Rowbotham). Regardless of the results of electric tests, I would never postpone surgical intervention for more than two months; Rowbotham, not longer than three months.

This mode of action can be adopted without any risk of impairing the hearing in (*a*) transverse fractures of the temporal bone, because both vestibular and acoustic functions are lost, and (*b*) in longitudinal fractures where the nerve is injured in the pyramidal and vertical segments, because exploration can be done without injuring the contents of the middle ear.

If, however, the nerve is injured in the horizontal segment, there is a risk of destroying the middle ear. In some cases, even in this site, the nerve may be repaired without damaging the middle ear (Maxwell and Magielski, Kettel), but, on the other hand, it may be necessary to do a radical mastoidectomy in order to repair the nerve. Needless to say, the patient should be told in advance.

II. *Palsies Occurring After an Interval of Freedom.*—As the continuity of the nerve is preserved, the prognosis is generally fair. If, however, serial electromyograms, which should be started early, show a severe interference with conduction, decompression and inspection should be seriously considered, just as in the immediate palsies, as outlined above, and as stated in a previous paper. Botman and Jongkees recently arrived at the same conclusions. When an electromyograph is not available, exploration should not be postponed for more than two months.

## Results Obtained by Surgical Intervention

### Review of the Literature

We may now consider the observations which have been made at the operating table, and the results which have been obtained by active intervention.

Cawthorne published a report of six cases in which he mentions finding "a severe fracture with fragmentation involving the inner tympanic wall and the facial nerve in its tympanic segment." From him I learned that in two cases the nerve was torn. In the first, a gap was bridged by an 8 mm. graft. In the second, the stumps, after being trimmed, were sealed together with concentrated plasma solution. In four cases the nerve was badly bruised. In all six cases there was return to function.

Farrior and Caldwell performed a decompression in a case in which, two months after the accident, there was still complete facial paralysis and mastoiditis. The fracture of the temporal bone had extended through the roof of the antrum, and sequestra were removed from a fistulous tract into the external auditory canal. In the infralabyrinthine area, a comminuted fracture was noted immediately below the posterior semicircular canal. Decompression of the vertical course of the facial nerve disclosed several small spicules of bone impinging upon the medial and posterior surfaces of the facial nerve, immediately below the horizontal semicircular canal. These spicules were removed. The underlying portion of the facial nerve was contused and edematous.

Behrman reported a case of transverse fracture with complete facial paralysis and destruction of the labyrinth. Two months after the paralysis he explored the nerve and observed a fracture through the ampulla of the lateral semicircular canal and the facial canal. The nerve was lacerated and the continuity broken. A graft was inserted, and facial movement was regained.

Botman and Jongkees reported five cases, in four of which "results of decompression of the nerve, constricted in the fracture and

often jammed in between the two shifted bone-masses, are remarkably good." In one case the nerve was severed, and nerve grafting was successfully performed. Torre has operated on two cases of facial paralysis with fracture of the petrous pyramid. In the first case the nerve was compressed by fragments near the bend at the posterior end of the lateral semicircular canal. Bloch and Aboulker report a case of longitudinal fracture and immediate palsy. Two months after occurrence a decompression was done, and below the aditus the canal was soft and the nerve red. Quick recovery followed. The authors are uncertain whether this was because of or in spite of the operation!

Ombredanne found a mobile sequestrum near the point of the facial ridge four and one-half months after an accident. Four-fifths of the thickness of the nerve was cut at this point. The gap was bridged by a graft, with ensuing good result.

Aboulker operated on a man who for three years had had facial palsy caused by a gunshot. X-rays showed a fracture of the temporal bone involving the region of the oval window. The nerve was atrophic but not severed. The sheath was split, but no improvement had resulted one and one-half years after the operation.

Lemoyne operated on a case with a fracture near the second bend of the nerve and found a hematoma in the nerve. After splitting of the sheath, rapid recovery followed.

Recently Maxwell and Magielski reported three cases of complete peripheral facial paralysis due to fractures of the middle fossa. In one case bone fragments compressing the pyramidal segment of the nerve were removed, and the nerve was decompressed from the geniculate ganglion to the stylomastoid foramen, without removing the ossicles. A marked improvement followed five days after the operation and was fairly satisfactory two months later. The postoperative hearing was nearly normal.

The second patient had multiple cranial nerve palsies and a delayed facial paralysis on the left side. Multiple fracture lines were seen on both sides; on the left side one extended from the temporoparietal area into the mastoid. Five weeks later decompression was done, as the palsy was complete and electromyography indicated complete denervation. The fracture line passed through the pyramidal segment of the facial canal, and here the nerve was grossly edematous and bulged through the opened sheath. Excellent recovery followed.

In the third case the fracture line passed through the horizontal semicircular canal and the facial canal. The nerve was severed just distal to the geniculate ganglion, and a nerve graft was inserted, with ensuing good result.

### Own Cases

Included in this series are 16 patients, 1 of them with an immediate bilateral palsy, i. e., 16 patients but 17 cases. Ten patients were male (8-65 years of age) and six female (27-65 years of age). In 14 of these cases the skull fracture was due to a traffic accident, and in the remaining 3 cases the patients lost their balance for various reasons, hitting the back of the skull in falling.

In 16 cases the fracture was of the longitudinal type and in 1 of the transverse variety, thus confirming the fact that the longitudinal fracture is by far the commoner.

All the patients were initially treated in various surgical departments and were later referred to Frederiksborg County Hospital on account of persistent facial palsy. One patient was referred a few days after injury, one 1 month, nine 2-4 months, and five 7, 9, 10, 16, and 20 months, respectively, after the accident. As very little has been written on the surgical treatment of palsies due to skull fractures, and since the general belief is that they all should be treated conservatively, the following cases will be described in some detail.

*Clinical Symptoms.*—1. Symptoms Arising in Direct Conjunction with Injury: The cases may be divided into two groups.

Group 1. In 12 patients the main symptoms were initially identical: (*a*) loss of consciousness, lasting from a few hours up

to a week; (*b*) bleeding from one or both ears; (c) impaired hearing, and (*d*) complete peripheral facial palsy, apparently arising in direct conjunction with the accident.

In three of these cases the palsy was bilateral, but only in one of them did the palsy appear on both sides in immediate conjunction with the accident. In the other two cases the palsy was noted immediately after injury on one side and after an interval of a few days on the other. The delayed palsies disappeared spontaneously later. In these three patients paralysis of one or both abducens nerves was also noted.

Group 2. Four of the patients, all with an immediate and complete peripheral facial palsy on one side, did not lose consciousness. In three of them there was bleeding from the homolateral ear, while in the fourth palsy was the only clinical symptom.

*X-Ray Examination.*—Fifteen of the patients underwent x-ray examination shortly after the accident, and in fourteen of them fracture lines were seen. These were generally single but occasionally multiple, and started in the parietotemporal area, running downward and forward, and generally involving the mastoid process near the second bend of the facial canal, at the posterior end of the lateral semicircular canal.

*Otological Examination.*—All the patients complained of impaired hearing on the side involved, but, from lack of otological assistance in some of the hospitals where the patients were initially treated, detailed information on some of the cases is not available. In those patients seen by an otologist, bleeding in the external meatus, a rupture of the drum, or a hematotympanum was noted.

In only one case (No. 12), in which the hearing disappeared completely and immediately, was a caloric test done, which proved to be negative. This is the only case of a transverse fracture in this series.

2. Symptoms on Admission at the Time of Repair of the Facial Nerve: As already stated, most of the patients were referred after observation for some time, in most cases after two to four months. At this time all cerebral symptoms had disappeared.

The peripheral facial palsy was complete in 10 patients (in 1 of them on both sides), while in 6 patients very faint concentrations at the corner of the mouth were the only visible signs of facial nerve function.

*Electrotest.*—The galvanic reaction was positive in all the patients, while the faradic reaction was negative in all cases in which the palsy was complete.

Electromyography was done in only five cases, since the apparatus is not available at F. C. H. and we have to send patients 25 miles away for examination. In four of these cases a complete block of conduction with denervation potentials was registered, while in the fifth case severe interference with conduction only was found.

I personally have performed decompression on 16 patients, in 1 of them on both sides. In 14 of the cases definite alterations were found in the bone and the nerve, and in 10 cases these were severe. In three cases nothing macroscopically abnormal was found. In no case was the nerve severed.

According to the site of lesion, the cases may be divided into three groups. To avoid repetition, it should be stressed (1) that except where stated, all the patients were unconscious for one to seven days in conjunction with the accident; (2) that a complete peripheral facial palsy occurred, to the best of our knowledge, in direct conjunction with the injury, and was still complete when decompression was done; (3) that the faradic reaction was negative but the galvanic positive; (4) that there had been bleeding from the homolateral ear, and (5) that the hearing was impaired at least initially. In Case 12 the fracture was transverse, while in all the other cases fracture was of the longitudinal type.

*Group 1. Injuries to the Vertical Segment of the Facial Canal and Nerve.*—This group comprises four cases.

CASE 1.—Woman, 44 years of age. Roentgenograms showed no signs of fracture of the temporal bone. Ten months after the accident the palsy

was still complete. On decompression of the vertical segment of the nerve, no fracture lines were observed. The middle third of the nerve was red and swollen, but above and below this segment it looked normal. As the hearing was good, the horizontal segment of the nerve was not uncovered. At reexamination nine months later there was no improvement, and, as her left eye was watering, which troubled her considerably, a partial lateral tarsorrhaphy was done.

One year nine months after decompression a considerable improvement was noted. She looked normal with the face at rest, and she could screw up her eye, smile, and laugh to a limited degree, but not wrinkle her forehead.

Comment: When no movements could be noted after 10 months of observation, I considered an explorative decompression justified. The palsy may have been due to a hemorrhage in the facial canal, where the nerve was red and swollen. A good improvement followed. It is a matter of conjecture whether this was in spite of or on account of decompression.

Case 2.—Man, 19 years of age. He was referred to F. C. H. four months after the accident. It was impossible to tell whether the palsy had occurred in direct conjunction with the accident or after an interval of freedom. As faint contractions were noted, the faradic reaction was positive, and no signs of fracture were seen on the roentgenograms, prolonged conservative treatment was advised.

Three years three months after the accident he was once again admitted to F. C. H. He could close his eye, but only faintly, and his mouth was practically immobile on the affected side. His forehead was completely immobile. On decompression, a necrosis of the wall of the facial canal was observed in the middle third of the vertical segment. The nerve looked somewhat atrophic at this point. The sheath was split.

At reexamination three months later the mobility of the mouth had improved slightly. In a letter nine months after decompression, he stated that only a very limited improvement had followed.

Comment: Since after three years three months of observation the palsy remained severe, a decompression was considered justified.

No improvement followed, probably because of intraneural scar formations in the vertical segment of the nerve. I should have operated when he was first referred to me, and, failing to do this, I should have at least resected the atrophic part of the nerve, which probably consisted of connective tissue, and inserted a graft when he was referred the second time.

Case 3.—Woman, 39 years of age. As a result of an accident in 1946, she had bleeding from the right ear and ecchymoses on the right mastoid process, with complete facial paralysis. Several weeks afterward, movements began at the corner of her mouth, but the improvement soon stopped. Nine months later she was referred to F. C. H. She was able to smile a little, but could not close her right eye or wrinkle her forehead. However, as the faradic current evoked strong contractions around her eye and of her forehead, it was considered justifiable to try to improve the spontaneous function by a decompression. In the cells between the sigmoid sinus and the posterior osseous wall, stale blood and cellular destruction were found, obviously remnants of a fracture. The facial canal, however, was macroscopically normal, and the nerve did not bulge after the sheath was split.

On reexamination three years later she looked normal with the face at rest. She could close her eye, and the movements of her mouth were as before the decompression. Her forehead was immobile.

Comment: It is fair to assume that the improvement was due to decompression. For a long time before the operation there had been no improvement at all, but it started in direct conjunction with the operation, probably because pressure had been relieved.

Case 4.—Woman, 28 years of age. Roentgenograms revealed a fracture of the right temporal bone, involving the posterior section of the petrous part. She was admitted to F. C. H. 16 months after the accident. Only faint contractions at the corner of her mouth could be noted, and for many months there had been no improvement.

On decompression no fracture lines were seen. The vertical part of the facial canal was opened, and on slitting of the nerve sheath the nerve bulged outward.

At reexamination 18 months later a considerable improvement was noted. She looked normal with the face at rest, and was able to screw up her eye and smile and laugh, though not as much as on the other side. Her forehead was immobile. (Illustrations were published previously.)

Comment: As there had been no improvement for a long time before the operation, and as mobility started in direct conjunction with decompression, it is fair to assume that

the palsy was due to pressure within the facial canal, the neurofibrils bulging outward on decompression.

In these four cases only limited alterations were noted in the vertical segment of the facial canal.

Case 1: Middle third of the nerve red and swollen

Case 2: Limited necrosis of the wall of the facial canal and the nerve atrophic

Case 3: Stale blood and cellular destruction between the sigmoid sinus and the posterior osseous wall, whereas the facial canal and nerve looked normal

Case 4: Edema of the vertical segment of the nerve

A good improvement resulted from the operation in three of the cases, operated on 9, 10, and 16 months, respectively, after the accident, and in two of them it started in direct conjunction with decompression. In none of the cases, however, was a complete cure obtained, and a better result might have been expected if the operation had been done at an earlier date.

In one case (No. 2) no improvement followed, but it could hardly be expected, as the nerve looked atrophic. The patient was not operated on until three years three months after the accident (in this instance also at a date much too late), and then a graft should have been inserted to replace the atrophic part of the nerve.

*Group 2. Injuries at the Second Bend of the Nerve* (At the posterior end of the lateral semicircular canal).—This group comprises seven cases.

Case 5.—Woman, 27 years of age. Bilateral facial palsy due to a fracture of the skull. On the right side the palsy did not appear till several days after the accident and it disappeared spontaneously and completely within three weeks; on the left side the palsy was noted in immediate conjunction with the accident and was complete from the start. Roentgenograms revealed fracture lines of the squamous part of the temporal bone, involving the mastoid process. Palsy was complete, and electromyography showed no function of m. frontalis, severe interference with conduction of m. orbicularis oculi et oris, and no function of m. triangularis, with denervation potentials in all three. In direct conjunction with the accident ecchymoses were seen on the left mastoid process, and the external acoustic meatus was reduced to a slit by bleeding between the posterior membranous and osseous walls. There was blood in the meatus. Two months later the meatus was practically normal, and the drum looked dull.

Decompression was done on the left side one and one-half months after the accident. The fracture was seen passing from the squamous part of the temporal bone through the posterior osseous wall of the external auditory meatus near the suprameatal spine of Henle. The posterior wall was fractured into several pieces, which had torn the nerve sheath near the second bend of the nerve.

On reexamination seven months later the palsy had disappeared almost completely on both sides (Fig. 4).

Comment: The indications were clear: complete palsy (Fig. 4) six weeks after the accident, a fracture involving the mastoid process, and almost complete block of conduction. Almost complete recovery, due to decompression, followed.

Case 6.—Man, 41 years of age. A fracture of the temporal bone was seen on roentgenograms, involving the petrous part near the point of the facial ridge. Decompression was done seven months after the accident. One line of fracture passed through the posterior wall near the suprameatal spine of Henle, and another through the facial canal in front of the ampulla of the lateral semicircular canal. The incus was dislodged into the middle ear, which was filled with scar tissue. The facial canal was not fractured. On decompression the horizontal segment of the nerve was seen to be red and enormously swollen, whereas the vertical segment looked normal.

Reexamination four and one-half years later: a partial tarsorrhaphy had been done shortly after the accident. The face at rest looked almost symmetrical. The patient could smile and laugh to some extent but not screw up her eye or wrinkle forehead.

Comment: As after seven months of observation there was practically no progress except for very faint contractions at the corner of the mouth, a decompression was considered necessary. From my point of view the result was unsatisfactory, but, fortunately, the patient was satisfied.

Extra- and intraneural bleeding with subsequent degeneration of axons is probably the cause of the palsy. A better result might have been expected if decompression had been done at an earlier date.

Fig. 4.—*A*, before operation. *B-D*, decompression on the left side was performed one and one-half months after the accident. Pictures *B-D* taken seven months after the operation.

Case 7.—Man, 65 years of age. Multiple fracture lines involving the petrous part of the temporal bone were seen on roentgenograms. He was unable to wrinkle his forehead or close his eye, and only faint contractions could be detected near the corner of the mouth.

Decompression was done four months after the accident. The posterior wall of the external auditory meatus was broken into several pieces. At its second bend, the nerve was dislodged, and heavily compressed, red, and very swollen. The middle ear was filled with scar tissue and remnants of blood. The horizontal part of the nerve looked normal when uncovered. Recovery started soon after decompression.

On reexamination, four years later, the patient looked practically normal, was able to screw up the eye, smile, and laugh, though not fully. He could not wrinkle the forehead.

Comment: The indications were clear: A practically complete conduction block after four months of observation, and fractures involving the petrous part of the temporal bone. The palsy was due to compression of the nerve, and extra- and intraneural bleedings. It is fair to assume that the recovery, which started in direct conjunction with decompression, is due to the surgical intervention.

Case 8. (See also Case 16).—Man 34 years of age. This case of bilateral palsy has been reported in detail previously (Arch. Otolaryng. 51:25, 1950). Right ear: Roentgenograms showed multiple fracture lines at the base of the petrous part of the temporal bone. Decompression was done nine weeks after the accident. A fracture

was noted through the tegmen tympani and the medial wall of the aditus ad antrum, involving the facial canal at the second bend. The fragments were dislocated, and the nerve heavily compressed but not severed. The incus was displaced into the middle ear, pushing the malleus outward. The nerve was uncovered, and it was seen that between the geniculate ganglion and the place of compression at the bend, intraneural hemorrhage had taken place, whereas the vertical segment looked normal. Rapid recovery took place.

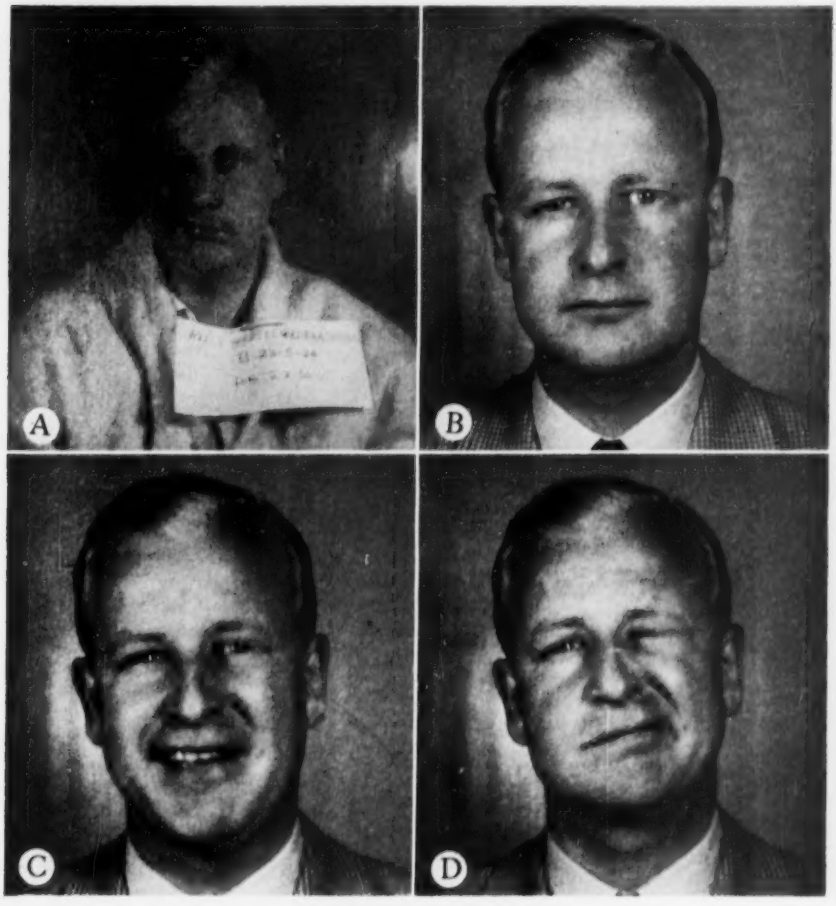
Reexamination seven months later: A picture of the result has been published previously. The face looked completely normal, and the only sequel of the palsy was that the smile was somewhat stiff.

Comment: The indication was clear: Complete palsy after nine weeks of observation and a fracture involving the petrous bone, with dislocation and compression of the nerve. The beneficial effect of the operation is obvious.

CASE 9.—Boy, 11 years of age. Complete facial palsy. Roentgenograms showed a fracture line in the squama of the temporal bone passing through the posterior wall of the external auditory meatus near the spine of Henle. Electromyography showed a complete interruption of conduction, and there were fibrillation potentials.

At decompression two months after the accident, the fracture corresponding to the roentgenograms was seen passing through the mastoid process and the facial canal near the second bend of the nerve. The fragments were dislodged, and the nerve heavily compressed, red, and swollen.

Fig. 5.—*A*, immediate and complete facial palsy on the left side due to fracture of the temporal bone. (The film was unfortunately damaged in developing.) *B-D*, five months after decompression.



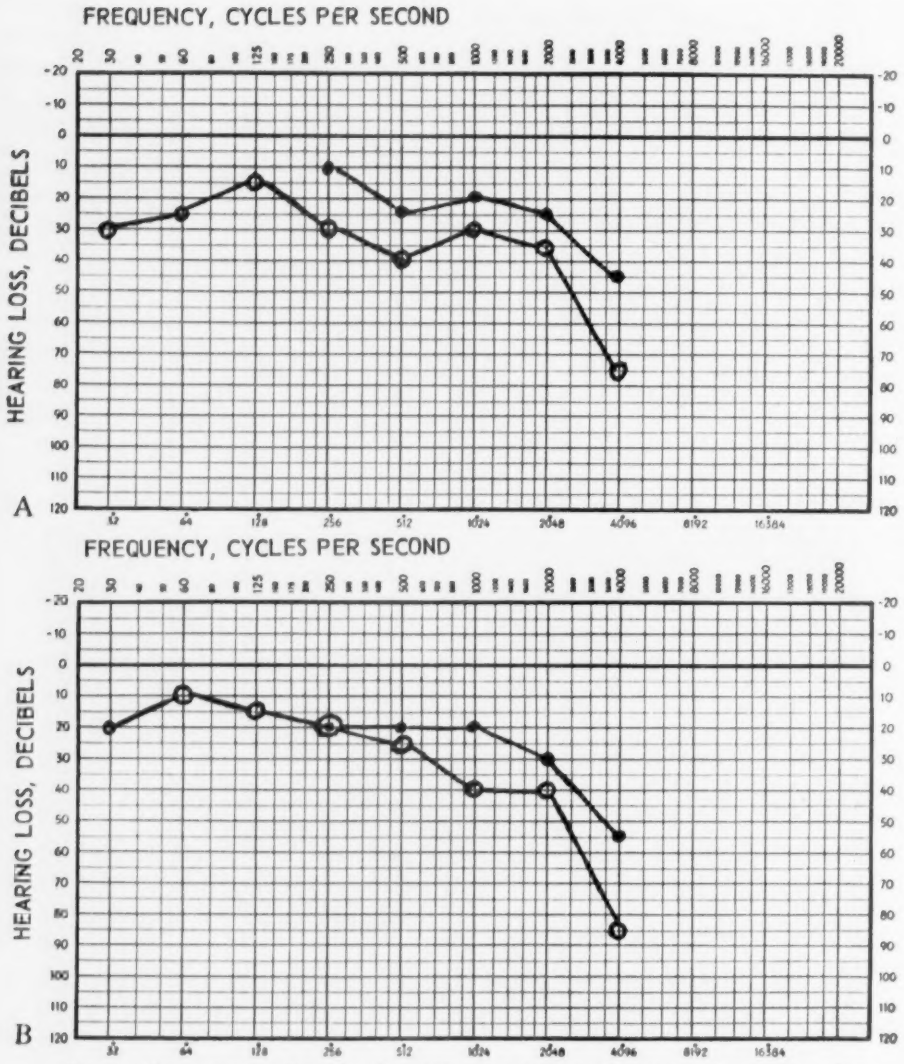



Fig. 6. (Same case as Fig. 5).—*A*, preoperative hearing. *B*, hearing five months after decompression, in which the facial nerve was decompressed as far as the geniculate ganglion without touching the ossicles. There is practically no loss of hearing. ——. bone conduction; o——o air conduction.

Remnants of blood and scar tissue filled the tympanic cavity. After decompression of the horizontal part of the nerve this segment looked normal.

On reexamination, two and one-half years later, the boy looked quite normal with the face at rest. Good, but not complete, function on the right side.

Comment: The indication was clear: After two months of observation there was massive palsy, complete block of conduction (electromyography), and a fracture involving the facial canal. The palsy was due to compression and contusion of the nerve. A clinically satisfactory result was obtained with decompression.

Case 10.—Man, 31 years of age. Roentgenograms showed a fracture line of the left temporal bone; the palsy was massive, and electromyography showed complete conduction block and denervation potentials.

On decompression two months later, the fracture was seen passing through the posterior osseous wall near the spine of Henle into the facial canal. A loose triangular fragment was compressing the nerve, which was contused at this point, and swollen. It was uncovered downward and upward as far as the geniculate ganglion, without dislocation of the ossicles (Fig. 5).

On reexamination five months later the patient looked normal with the face at rest; he could screw his eye up and smile even if not to full extent, but he is still improving. The preoperative hearing is practically unimpaired (Fig. 6).

Comment: The indication was clear. The palsy was obviously due to a fracture through the facial canal near the second bend, with a loose fragment compressing the nerve. The beneficial effect of decompression is obvious.

Case 11.—Man, 34 years of age. Roentgenograms showed a fracture line running from the squama of the temporal bone into the posterior osseous wall near the spine of Henle. It could be seen by otoscopy. Electromyography showed complete conduction block, with denervation potentials.

On decompression three months after the accident, the fracture line was seen corresponding to the roentgenograms, passing into the facial canal at the second bend. Bone spicules were pressed into the nerve sheath, which was torn, and the nerve was heavily swollen. Without dislodging the ossicles the horizontal part of the nerve was decompressed. Faint movements started in direct conjunction with decompression, and the preoperative hearing was not impaired.

Comment: The indication for decompression is clear. Four months after the operation the palsy was not noted with the face at rest; he could smile but not to full extent, and screw up his eye.

In the seven cases described above we are dealing with five patients with a complete peripheral facial palsy, and two in whom only very faint contractions at the corner of the mouth could be noted on close observation, four and seven months after the accident. Roentgenograms revealed fractures of the temporal bone involving the petrous part, and on decompression they were seen in each case passing through the posterior osseous wall near the spine of Henle. In one case (No. 6) there was an additional fracture in front of the cochleariform process. In all cases the nerve was compressed and heavily injured but never severed.

The indications for operation were clear. After one and one-half to seven months of observation there was in each case a complete (five cases) or practically complete (two cases, observed during four and seven months) peripheral facial palsy, and on roentgenograms a fracture involving the petrous part of the temporal bone was seen.

One patient (Case 6) considered the result satisfactory, though I did not.

In the other six cases the patients looked normal with the face at rest, could screw up the eye and smile and laugh to some extent, whereas the forehead was immobile. In none of the cases did the palsy disappear completely; some traces of it, even if often faint, remained.

*Group 3. Injuries to the Horizontal Segment of the Facial Canal and Nerve.*—This group comprises three cases.

Case 12.—Boy, 8 years of age. In this case the fracture was transverse, and cochlear and vestibular function were immediately destroyed. On roentgenograms a fracture through the labyrinth could be seen. The palsy was complete.

On decompression two months after the accident, the fracture through the labyrinth was clearly visible, but the facial canal was not involved. The horizontal and vertical segments of the nerve were uncovered, it was seen that while the horizontal was red and swollen, the vertical looked normal. The nerve was not severed. Spontaneous movements started in direct conjunction with decompression.

On reexamination nine months later the face was normal at rest; he could screw his eye up and smile and laugh, though not to the full extent, but he could not wrinkle his forehead.

Comment: The indication was clear: complete palsy after two months of observation, and destruction of the labyrinth. The palsy was due to an intracanalicular hemorrhage in the horizontal segment. A clinically satisfactory result was obtained, probably due to decompression.

Case 13.—Man 34 years of age. Roentgenograms revealed a fracture of the temporal bone involving the petrous part. The palsy was complete.

On decompression two months after the accident, one fracture line was seen passing through the superior wall of the external auditory meatus, and small fragments were found in the epitympanic recess. Another fracture line was seen to pass through the facial canal in front of the cochleariform process. The nerve was denuded, and bone splinters were impinging upon the sheath, but continuity was not broken. The horizontal segment of the nerve was red and heavily swollen, while the vertical looked normal. Granulations and scar tissue filled the tympanic cavity.

Reexamination three years later. He looked normal with the face at rest, could screw the eye up and smile and laugh, though not to full extent, and could not wrinkle the forehead.

Comment: The indication was clear: after two months of observation there was a massive palsy and a fracture involving the petrous part of the temporal bone. A clinically satisfactory result was obtained, due to the operation.

Case 14.—Woman, 31 years of age. Roentgenograms revealed a fracture of the temporal bone passing from the squama into the petrous part between the geniculate ganglion and the second bend of the nerve. Admitted to F. C. H. 20 months after the accident. There was a contracture on the left side, but she could move this side partially.

On decompression the fracture line was seen passing through the horizontal segment of the facial canal, and a bone splinter was impinging upon the nerve. Apart from this the nerve looked normal. The mobility present before the operation was not impaired.

Reexamination has not taken place, as she lives abroad.

Comment: The indications were equivocal. The patient could move the affected side partially, but a contracture was present. Being an actress, she insisted upon everything being done which might possibly improve her condition. As roentgenograms showed a fracture line through the horizontal part of the facial canal, and as I have seen cases of incomplete recovery improve after decompression, and on several occasions have noted a contracture disappear after decompression, I decided on operation, having explained the chances and risks. I succeeded in performing the operation without dislocating the ossicles, and the hearing was not impaired.

I have not seen the patient since, but from letters I have learned that there had been no improvement and that she was dissatisfied.

In two of these three cases the indication for operation was clear: After two months of observation there was a complete peripheral palsy, and a fracture involving the petrous part of the temporal bone.

In the last case (No. 14) I felt justified in doing a decompression as outlined above, but the indication was open to discussion.

A clinically satisfactory result was obtained in the first two of the cases. The last patient, who was not reexamined, wrote that the operation had been a disappointment.

In the following last three cases nothing abnormal was found at operation, and thus it is impossible to say to which of the three groups mentioned above they belong.

Case 15.—Man, 25 years of age. A depressed fracture of the right temporal bone was immediately cared for in a neurosurgical department. Moreover, there was a fracture of the orbit and jaw, and blood in the C. S. F. The hearing was impaired, but there was no bleeding from the ears. Admitted two months after the accident. There was a complete peripheral facial palsy on the left side, and electromyograms showed complete conduction block. Roentgenograms showed no fracture lines of the left temporal bone, and the hearing was normal. On decompression of the vertical segment of the nerve, nothing abnormal was found. On reexamination two years later he looked normal with the face at rest, apart from the right eye lying somewhat deeper in its socket (fracture of the orbit). He could wrinkle the forehead a little, screw up the eye and smile, and laugh to some extent.

Comment: Nothing abnormal was found in the vertical segment of the nerve, and as the hearing was normal the operation was not carried as far as the geniculate ganglion. This operation was probably unjustified, and the palsy may have been due to a hemorrhage in the internal auditory meatus. It should be remembered that there was blood in the C. S. F. in conjunction with the accident. The improvement which followed probably took place in spite of the operation.

Case 16.—Boy, 9 years of age. No fracture lines were seen on roentgenograms. Electro-

encephalography and examination of the C. S. F. were normal. Three and one-half months after the accident the palsy was still complete, and a decompression was performed. Without dislocating the ossicles, the nerve was uncovered from the geniculate ganglion to the stylomastoid foramen, but nothing macroscopically abnormal was found. On reexamination four years later he looked completely normal, was able to screw up the eye and smile and laugh, though not to full extent, but did not wrinkle the forehead.

Comment: As improvement started immediately after decompression, it is fair to assume that the palsy was due to pressure within the facial canal, which was relieved by decompression.

Case 17.—Man, 34 years of age. This patient had a bilateral complete facial palsy and paralysis of both abducens nerves in direct conjunction with the accident. Case 8 deals with the right side. Two and one-half months after the accident the palsy was still complete on the left side, and a decompression performed.

No fracture lines were seen on roentgenograms. Partial reaction of degeneration was present. The nerve was uncovered from the stylomastoid foramen to the second bend, but nothing macroscopically abnormal was found. On reexamination seven months later the palsy had almost completely disappeared, but the smile was a little stiff. He could move the eyes freely. Pictures have been published previously.

Comment: It should be remembered that this patient had a bilateral complete facial palsy in direct conjunction with the operation, and a bilateral paralysis of both abducens nerves. The reason for the palsy of the right facial nerve was compression between dislodged fragments (Case 8). As mobility on the left side started on the day of decompression, it is fair to assume that a pressure upon the nerve in the facial canal was the cause of the palsy, and not intracranial bleeding, which might have been suspected.

In these three cases nothing macroscopically abnormal was found on operation. Nevertheless, improvement in two of these cases started in direct conjunction with decompression, perhaps indicating that a pressure upon the nerve was lifted. In one case (No. 15) decompression probably was unjustified, and the improvement that followed may have taken place in spite of the operation.

## Summary

From a review of the case records of 16 patients, one of them with a bilateral palsy (Cases No. 8 and 17) the following observations have been made.

In 16 cases the fracture of the temporal bone was longitudinal and in one transverse. In 14 out of the 15 cases submitted to x-ray shortly after the accident, the fractures were clearly visible, generally starting in the parietotemporal area and running downward and forward into the petrous part.

All the palsies belonged to the peripheral type. To the best of our knowledge, they started in direct conjunction with the accident, but it should be remembered that a facial palsy in a seriously injured patient may be overlooked for a short time, and the possibility that a few of the palsies may have arisen after a short interval of freedom cannot be excluded.

The patients were admitted to E. C. H. and operated upon 1½ to 20 months after the accident. In 11 cases the palsy was still complete on admission. In six only faint contractions at the corner of the mouth could be noted.

The indication for operation was clear in cases of complete palsy, with a fracture involving the petrous part of the temporal bone, and complete or severe interference with conduction shown by electrical tests. It was less clear in cases in which minor contractions were noted (which excludes a complete severance of the nerve), but where no progress had been noted during months of observation. However from experience of similar cases of Bell's ischemic facial palsy we know that further progress may eventually be gained by decompression. That this also applies to palsies due to fractures of the temporal bone is illustrated by Case 3.

The pathological findings varied. In three cases nothing macroscopically abnormal was noted at the operation. In 14 the pathological

findings ranged from edema of the nerve with no signs of fracture to nerves heavily compressed and injured by dislocated fragments. In none of my patients was the nerve severed, but Maxwell and Magielski, Cawthorne, Behrman, and Botman and Jongkees have reported such cases.

The nerve may be injured in the horizontal as well as the vertical segment, but most frequently at their junction at the second bend of the nerve, the pyramidal segment, through a fracture involving the facial canal at this point.

The results following decompression are as follows:

Fourteen patients looked normal with the face at rest; twelve of them could screw up the eye and smile and laugh, even if not to full extent, and in one case these movements were only faint. In one patient (Case 6, operated upon four and one-half years after injury) a partial lateral tarsorrhaphy had been done, and only slight contractions at the corner of the mouth were noted.

One patient has not been reexamined, but from letters I learn that the operation has been a disappointment to her.

#### Comment

It will immediately be seen that in not a single case was a complete cure obtained, by which is meant that not a single trace of the palsy was left. Clinically the results are quite satisfactory, as 10 patients had a complete palsy, 1 of them on both sides, and 6 an almost complete palsy (except for faint contractions at the corner of the mouth) months before the operation, whereas on reexamination of 15 of the patients 14 looked completely normal with the face at rest, and 13 could close the eyes and smile more or less normally. As far as I can judge from the available reports, the results are closely similar to those obtained by other surgeons. One patient has not been reexamined.

Have these results been obtained on account of or in spite of the operation? In cases where the nerve had been compressed and even dislodged by fragments, the connection between operation and results is obvious. In cases where edema or intracanalicular bleeding was observed, the patient might have improved after further delay through resorption of fluids. But, in my view, it is fair to assume that decompression has hastened recovery, especially when improvement started in direct conjunction with surgery, or where the spontaneous improvements had long ago stopped far short of complete recovery but started again after decompression.

In one of the cases (No. 15) I believe that the patient improved in spite of the operation; in one (No. 1) the indication may be open to question.

Could better results have been expected? Under the present conditions I believe not, because the majority were operated on at a rather late date. In cases in which the nerve is compressed between displaced fragments, it is obvious that the earlier it is freed, the better the chances of recovery. I think that this line of reasoning also applies to cases in which the palsy is due to pressure within the facial canal, which in itself does not abolish conduction but interferes with the nutrition of the nerve, leading to degeneration.

In closing, I should like to repeat: surgery is indicated as soon as possible in cases where severe interference with conduction in an accessible site can be diagnosed. Concerning palsies following facial lacerations, Maxwell said: "Undue delay called 'watchful waiting to determine whether or not the nerve has been injured permanently' causes disaster, and may condemn the patient to a life of embarrassing deformity."

This point of view could not be more clearly expressed. I believe it is absolutely correct and that it is equally valid in cases of facial palsy due to fractures of the temporal bone.

### III. Palsies Resulting from Facial Lacerations

Facial palsy may be due to lacerations of the face resulting from a variety of acci-

dents. "It has definitely increased as a result of the nature of modern war." (Lathrop).

*Indications.*—Having treated only one case of this nature, I have no personal experience of the field. Lathrop and Maxwell, on the other hand, have treated numerous cases with great success.

The nerve should be repaired whenever possible, and I agree completely with Maxwell when he states:

> If the patient's general condition permits of a prolonged surgical procedure, the optimum time to repair a damaged facial nerve is at the time of primary repair of the facial laceration. Late repair is sometimes necessary, but the inflammatory changes in the parotid gland render the identification of small branches of the nerve more difficult. This can be accomplished in many instances, however, to the sincere satisfaction of the patient.
>
> . . . If the facial paralysis is noted immediately after the laceration, and usually it will be, exploration to determine the exact type of injury is indicated. If for one reason or another exploration has been delayed, it must be remembered that the longer the delay has been, the poorer will be the results of repair. Some fibrosis of paralyzed facial muscles occurs in a year; after this the change is more rapidly progressive, militating against a good functional result from any type of nerve repair.

Lathrop is in complete accordance with Maxwell, and so am I.

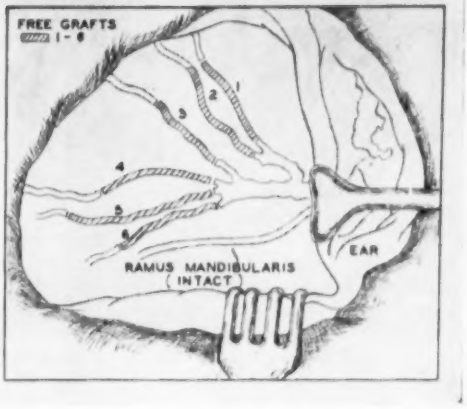
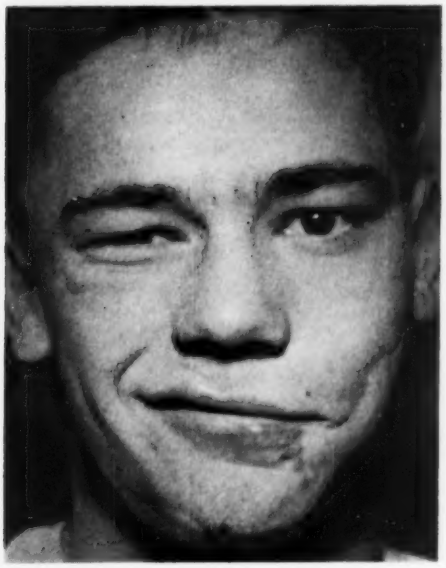



Fig. 8.—Repair was undertaken one month after the accident. Explanatory diagram showing multiple nerve grafts sutured in place. (From Tr. Am. Acad. Ophth. 58:733-740, 1954.)

Fig. 7.—This and the four following pictures are published by courtesy of Dr. J. H. Maxwell and the *Transactions of the American Academy of Ophthalmology and Otolaryngology*. I consider it the most skillful repair of a facial nerve, performed under extremely difficult conditions I ever saw.

The patient had suffered a severe laceration of the left side of the face in an automobile accident. There was no function of any of the facial muscles above the lower lip. (From Tr. Am. Acad. Ophth. 58:733-740, 1954.)



Woman 21 years of age.

Shot in the neck by the Nazis during the German occupation, just as in Schuerman's case. The bullet had traveled along the base of the skull, and the exit was found in the left cheek. A complete palsy occurred immediately, and suppuration followed. She was referred for repair one and one-half years later. The proximal end of the nerve was isolated at the stylomastoid foramen, the distal just proximal to the division at the pes anserinus. It was freshened, and macroscopy of the removed piece of nerve showed that it had been transformed into dense connective tissue. A 20 mm. graft was inserted without result, and later plastic operations were performed.

Comment: Repair had been postponed much too long, and failure was due to the fact that the distal ramifications and the tissue of the cheek had been transformed into dense connective tissue, through which no neurofibrils could reach the denervated muscles.

If repair is undertaken immediately, excellent results can be obtained in facial lacerations, even if the nerve has been extensively

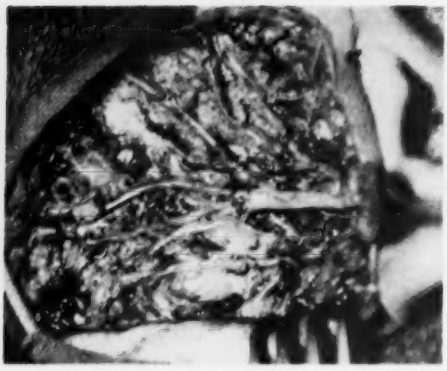


Fig. 9.—Photograph taken during the operation. (From Tr. Am. Acad. Ophth. 58:733-70, 1954.)

destroyed. By courtesy of Dr. Maxwell and the *Transactions of the American Academy of Ophthalmology and Otolaryngology*, a beautiful illustration of this is given (Figs. 7-11).

## Collected Results of Nerve Grafting and Nerve Suture in Seventy-Eight Cases

The total number of nerve graftings and nerve sutures which I have performed amounts to 78. The traumatic palsies, the principal theme of this paper, cover 71 repairs. The last seven patients have been operated upon on the following indications:

1. *Preoperative Facial Palsy in Otitis Media*

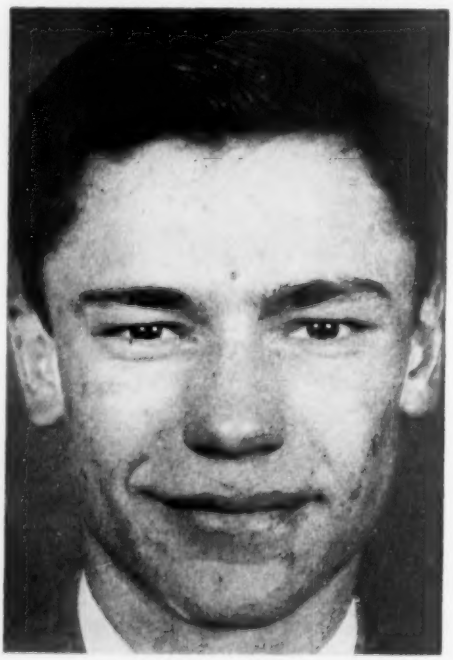
Man, 61 years of age. For years he had been suffering from a chronic otitis media, and for six months there had been a complete facial palsy. The hearing was destroyed, and there was negative response to the caloric test. The muscles showed partial reaction of degeneration. A radical mastoidectomy was done, and it was found that a large cholesteatoma had eroded the horizontal segment of the facial canal. The superficial three-fourths of the nerve fibers were missing, and only a tiny atrophic strand was observed in the depths of the lesion. This was preserved, and an end-to-side graft inserted, corresponding to the missing fibers. Complete failure resulted.

Comment: It is scarcely surprising that the result was so bad. There was partial reaction of degeneration, and, instead of removing the tiny atrophic strand which remained of the nerve, I preserved it. I have committed this error before. The right thing to do is to resect what looks like a tiny nerve strand, but is generally connective tissue. When a clear-cut of the nerve has been obtained at a point where it looks normal, a graft can be inserted.

Youth, 17 years of age. From the age of one year he had suffered from recurring otitis media on the left side. When he was 7 a complete peripheral palsy developed, and one year later he was referred to F. C. H., still with complete palsy. The left ear was dry, with a large central perforation. Whisper 4.00.

A decompression was done. The mastoid cells were necrotic, filled with a black fluid. The wall of the facial canal was very soft, and easily opened. The condition of the nerve is not recorded. I saw him again nine years later. He had been constantly treated with galvanization and massage, but only faint contractions could be noted at the side of the nose. He was unable to close the eye, and the mouth was completely immobile. He was examined electromyographically, and, as contractility was quite good, nerve grafting was suggested. From just distal to the geniculate ganglion to the pes anserinus, the nerve looked atrophic (microscopy: fibrous connective tissue), and a 48 mm. graft was

Fig. 10.—Result 12½ months after operation. (From Tr. Am. Acad. Ophth. 58:733-740, 1954.)



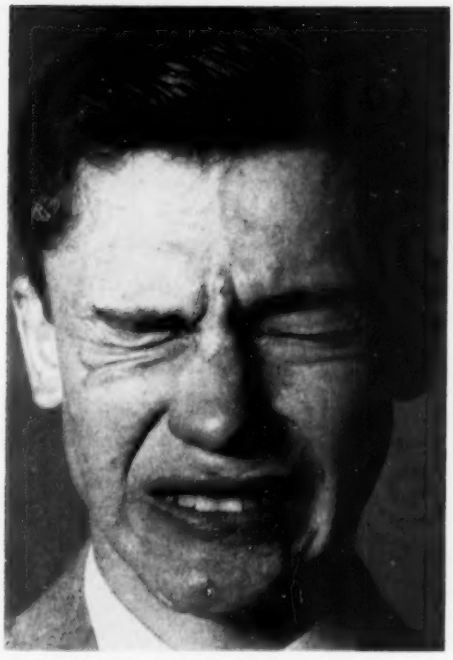


Fig. 11.—Result 12½ months after operation. It should be noted that there was some function of the frontalis muscle even if minimal. I have noted the same on six occasions, in contrast to a unanimous statement in the literature that this never happens after nerve grafting or nerve suture of the facial nerve. (From Tr. Am. Acad. Ophth. 58:733-740, 1954.)

inserted. Treatment is not finished, and the result remains to be seen.

Comment: In the majority of cases, a facial palsy in conjunction with acute otitis media will disappear spontaneously if the otitis is treated adequately. If the palsy remains complete, operation should be considered not later than two months after onset, or earlier if an electromyograph is available and severe interference with conduction can be demonstrated.

*2. Tumors of the facial nerve*

On two occasions I have performed decompression of the facial nerve on an erroneous diagnosis of Bell's ischemic palsy. In one case a neurinoma, and in the other a sarcoma was found on the descending vertical segment of the nerve. Both cases have been published in this periodical previously and reference is made to the earlier papers for detailed information.

Woman 32 years of age. The clinical picture was, as previously described, exactly that of a recurring Bell's palsy. On decompression a cavity larger than a hazelnut containing reddish-gray masses (microscopy: neurinoma) was found in the mastoid process, near the stylomastoid foramen. The wall of the facial canal was destroyed between the second bend of the nerve and the stylomastoid foramen, and the facial nerve had thus been exposed to view. The continuity of the superficial fibers was broken, and they were carefully placed in contact with one another. The deeper fibers looked uninjured. Eighteen months later there were no signs of returning mobility, and a second operation was performed. No connection could now be found between the proximal and the distal part of the facial nerve, and consequently a graft was inserted. Complete failure resulted, and plastic operations were later performed elsewhere.

Comment: The conditions for repair were good when I first saw the patient, but again I committed the error of preserving nerve fibers which looked viable. I should have resected the strand connecting the normal proximal and distal ends of the nerve and inserted a graft when I first saw the patient.

Woman, 57 years of age. As described previously, a reddish irregular solid tumor the size of a small hazelnut was found between the sigmoid sinus and the posterior osseous wall. It had destroyed the posterior osseous wall of the facial canal from a few millimeters below the second bend of the nerve to a few milimeters proximal to the stylomastoid foramen, expanding into the mastoid cells posteriorly. The tumor, a spindle-cell sarcoma, had completely infiltrated the stem of the nerve. It was resected, and a graft inserted.

Comment: On reexamination six years later there was no recurrence of the tumor. The angle of the mouth was somewhat drooping, even if it could be moved freely. She could not close the eye, and a partial lateral tarsorrhaphy had been done. Consequently this case is registered as clinically unsatisfactory, although as a matter of fact the patient is happy with the result (Fig. 12.)

Postoperatively she received x-ray therapy. It is possible that this treatment may have destroyed part of the graft and is responsible for the poor result.

Fig. 12.—*A*, complete facial palsy of the left facial nerve due to a sarcoma of the vertical segment. *B-D*, six years later. As the palsy is noted with the face at rest, this case is classified as clinically unsatisfactory. As a matter of fact the patient is more than happy with the result.

*3. Bell's Ischemic Palsy*

Among the cases of traumatic facial palsy is one of Bell's palsy (already described), where a decompression had been done elsewhere, the nerve being cut. I have, however, operated on three other cases of this condition in which nerve grafts were needed because of degeneration of the nerve, and not as a result of trauma.

Woman 30 years of age. This case has been described in detail previously. There is a general agreement that Bell's palsy is due to ischemia of the facial nerve. As a result of lack of blood supply, the nerve is paralyzed, and various alterations take place in it, as described in detail elsewhere. (Kettel) Moreover, I have been able to show that in some cases an aseptic necrosis of bone can be demonstrated in the mastoid process, findings which have been confirmed by others (Skoog, Hall, Flodgren, Sokcic). In this case the destruction of the mastoid cells was very extensive, the vertical part of the facial canal was destroyed, and the nerve was lying exposed to view. As a result of ischemia, necrosis of the nerve with complete interruption of continuity of the vertical segment had taken place. A nerve graft was inserted, and a good result was obtained.

Comment: This is a very extraordinary case of Bell's palsy. An ischemic necrosis of the mastoid cells has been observed on several occasions, but never before a necrosis

of the nerve with interruption of continuity. It is the first case of Bell's palsy in the literature in which a nerve from a living person was subjected to microscopy. This demonstrated conclusively that the violent alterations which were observed were due to vascular disturbances, while no signs of infection were found. A good result followed nerve grafting. Later, two more nerves which had been transformed into connective tissue were subjected to microscopy (See below).

Man 27 years of age. When he was 5 years old he acquired a complete Bell's palsy on the right side. He was treated by means of galvanization and massage for years, but he could only move the angle of the mouth very little, and not close the eye or wrinkle the forehead. When he was 25 years old, nerve grafting was attempted elsewhere. The vertical segment of the nerve was completely atrophic, but was not resected, and along this string three cable grafts were inserted without result.

Two years later he was referred to F. C. H. still with a complete palsy. As the response to the galvanic current was strong, thanks to intensive electrical treatment over the years, nerve grafting was done once more. Between the second bend of the nerve and a point just proximal to the pes anserinus, the nerve was transformed into an atrophic strand. (Microscopy: fibrous degeneration of the nerve). This was resected, and a 40 mm. graft inserted. Mobility started to return 14 months later. On reexamination two years after operation, the mouth was straight, and he could move it to a limited degree. However he could not close the eye, and a partial lateral tarsorrhaphy was done. The forehead was immobile.

Comment: When he was operated on the first time, the atrophic strand should have been resected, but it was erroneously preserved, just as I have done on some occasions. It is interesting to note that, thanks to continued conservative treatment, which had kept the muscles alive, a graft was successfully inserted 22 years after onset of the palsy.

Girl, 2 years of age. When she was 15 months old, a complete facial palsy occurred on the right side. As after six months of observation no signs of returning mobility were noted, a decompression was performed, but without any result. Two years three months after onset of the palsy, she was referred to F. C. H. From the second bend of the nerve to a point just outside the stylomastoid foramen, the nerve had been transformed into an atrophic strand, which was resected. (Microscopy: connective tissue, probably containing nerve fibers). A 15 mm. graft was inserted. One year later she looked normal with the face at rest; she could move the angle of the mouth a little, and could close the eye. She was still improving.

Bell's ischemic palsy is generally of a very benign nature, and is due to ischemia, a "dysregulation" of the vasa nervorum near the stylomastoid foramen. In the majority of cases the process is reversible, but not always. Three cases of a serious nature are reported. In one a necrosis of the nerve with interruption of continuity was observed; in the other two the nerve had been transformed into a strand of connective tissue. A graft was inserted in these three cases, and clinically satisfactory result were obtained in all of them.

### Review of Results

I have attempted 70 nerve graftings according to the technique of Ballance and Duel and 8 nerve sutures following the method of Bunnell. Six of the cases have been referred to me only recently. In 72 cases the treatment is finished. It must be borne in mind that not even the most successful operation will be able to restore function completely. The minimum requirement for a result to be classed as satisfactorily is that the palsy must not be noted with the face at rest, and that the patient can smile to some extent. In the majority of cases they could also screw up the eye, whereas only a few patients could wrinkle the forehead a little.

The material may be divided into two groups:

I. *Favorable Cases.*—These are cases in which no degeneration of the muscles had taken place at the time of repair and in which the operation was successfully performed. To this group belong 58 cases.

As a result of the operation in these 58 cases, all the patients looked normal with the face at rest; 6 patients could only smile; 40 patients could smile and screw up the eye; 6 patients could smile, screw up the eye, and wrinkle the forehead a little.

In six cases there was complete or partial failure.

In contrast to other surgeons experienced in facial nerve surgery I have for years maintained that even function of the frontal muscles may return after nerve grafting or nerve suture. I have noted it in six patients. It has now been confirmed by Maxwell. It should be remembered that the majority of the failures were in operations done before I started using magnification, and probably the coaptation was bad.

II. *Unfavorable Cases.*—These are cases in which the muscles had degenerated partially at the time of repair or in which I found insurmountable technical difficulties. To this group belong 14 patients. The operation in these cases was an attempt to obtain reinnervation. The issue was problematic beforehand, and the patients had been told in advance.

As previously explained, it is no wonder that the "results" in this group are so bad, as it was either quite impossible to isolate the peripheral stump of the facial nerve, or only a tiny strand of the peripheral ramifications was found. This means that as a matter of fact the operation in the majority of these cases could not be completed. In the rest of the cases partial degeneration of the muscles had taken place at the time of repair. However, there was nothing to lose and everything to gain.

The interval between damage and repair has varied widely. Many repairs have been done immediately after the palsy was noted, the majority with one-half year, and a few done years later.

The length of the graft has varied from 4 to 50 mm. Good results may be obtained by means of short (Fig. 13) as well as long grafts (Fig. 14).

The first signs of returning mobility were noted from 4 to 24 months after repair, on an average of 10 months, regardless of whether a long or a short graft or a nerve suture was used. That is easily understood, as the neurofibrils, regardless of the mode of repair, will travel from the proximal stump of the facial nerve through or along the graft into the denervated muscles.

A damaged facial nerve should be repaired as soon as possible but, as illustrated by my examples in the text, repair may be successfully done even years after the damage, provided that the site of injury is accessible and the operation skilfully performed under the microscope, and last, but not least, that the muscles have not degenerated.

Fig. 13.—Intratemporal nerve grafting, 10 mm. *A*, before. *B* and *C*, two and three-fourths years after the operation.

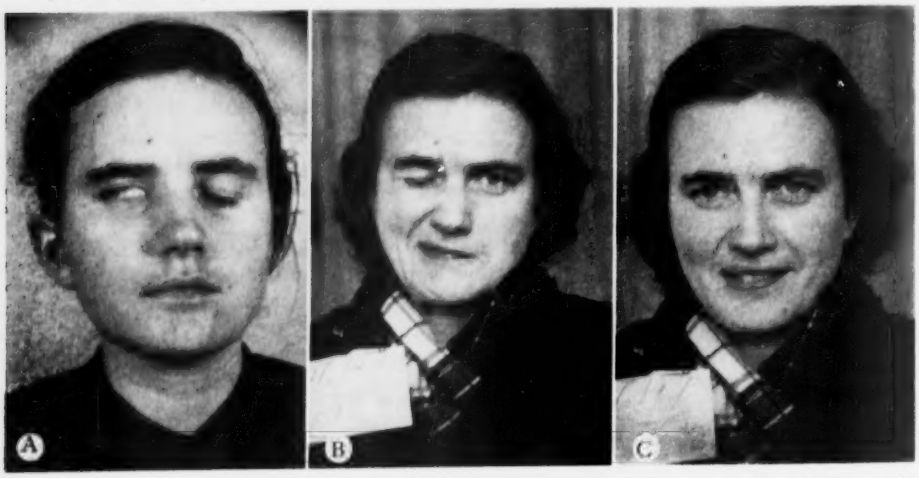


Fig. 14.—Intra- 4- extratemporal nerve grafting, 35 mm. *A*, before. *B–D*, two years after operation.

## Summary

In traumatic facial palsy all our endeavors must aim at restoring mimicry. The only way to reach this goal is to repair the nerve at the site of the lesion by decompression, nerve grafting, or nerve suture.

The prognosis of traumatic facial palsy is serious when the palsy occurs in direct conjunction with an operation or accident, because the continuity of the nerve is very often interrupted.

The prognosis becomes fair if the palsy occurs after an interval of freedom.

The traumatic palsies are divided into (1) postoperative cases, (2) palsies due to fractures of the temporal bone and (3) palsies due to lacerations of the face. My material comprises 121 patients, to which number

should be added 7 cases in which nerve grafting was performed on nontraumatic indications. The postoperative palsies may be due to intracranial operations, especially in removing a neurinoma of the acoustic nerve. I feel that our endeavors should aim at repairing the nerve at the site of injury, and that we should not content ourselves with an anastomosis operation, which never will restore mimicry. A report is given of 97 cases in which facial palsy occurred in direct conjunction with operations on the temporal bone. In 64 patients the continuity of the nerve was interrupted, and nerve grafting or nerve suture was performed. In 33 patients the nerve was damaged without being severed. Facial palsy may also be due to extratemporal operations outside the stylomastoid foramen and are especially seen following removal of a tumor of the parotic gland. This group comprises five cases.

The indications for repair of the nerve are discussed, and the results obtained under varying conditions are reported.

Traumatic facial palsy may also be due to fractures of the temporal bone. A detailed report of the case histories of 16 patients, one of them with a bilateral palsy, is given. The indications for surgical intervention upon the facial nerve are discussed and the results reported. A clinically satisfactory result was obtained in all the patients.

The facial nerve may be seriously injured by lacerations of the face and should be repaired as soon as possible. To this group only one patient belongs.

Included in the material are seven cases of nontraumatic origin, in which nerve grafting was needed (tumors of the facial nerve, preoperative otogenous palsies, Bell's ischemic palsy).

In all, nerve grafting and nerve suture have been attempted in 78 cases, and in 72 of these treatment is finished. The results may be divided into (1) favorable cases and (2) unfavorable cases.

1. Favorable cases are those in which no degeneration had taken place at the time of repair and in which the repair was successfully performed. To this group belong 58 cases, and in 52, or 90%, a clinically satisfactory result was obtained by repairing the nerve at the site of lesion. The reasons of the failures are briefly discussed.

2. Unfavorable cases are those in which either partial degeneration of the muscles at the time of repair had taken place or in which technical difficulties, to me insurmountable, were encountered. In the majority of these cases of long-standing palsies it was impossible to isolate the peripheral ramifications of the facial nerve in dense scar tissue of the cheek, and thus the operation could not be completed. This group comprises 14 patients.

If the facial nerve is injured it should be repaired as soon as possible. Repair, should however also be tried in cases of longer standing, provided the site of injury is accessible and the muscles of the face have not degenerated. There is everything to gain and nothing to lose. The results are most satisfactory.

Slotsgade 34.

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# Prevention of Adverse Effects of Streptomycin on the Ear

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How many years has it been since Waksman discovered streptomycin? And how many years has it been since the adverse after-effect of streptomycin on hearing ability became known?

Research in antibiotics having advanced, when will it be before an antibiotic without adverse after-effects is discovered. When I think of today it is difficult for me at times to put myself wholeheartedly into my work. But, sad to say, we who have direct contact with the patients are in the position of responsibility for eliminating, so far as possible, these adverse after-effects, hoping that the new antibiotic may be found in the near future. Until that day research must be continued with unfailing zeal. I have learned from a journal that new drugs have been discovered that are similar to streptomycin, without their adverse after-effects, but they are said to be in an experimental stage. I hope that the discovery of new drugs can be widely used in the near future and that the treatment which I am about to advocate will become obsolete or unnecessary.

Dr. Morita of Osaka University Hospital in an autopsy made a histological study of the inner ear of a man who had suffered adverse after-effects from streptomycin. He also has done similar research on animals. According to his research, the outer and inner hair cells and the tectorian membrane press against each other and form a straight line. If such a change occurs, it is shown histologically that any recovery may be said to be impossible.

I had previously thought that the adverse effect of streptomycin was due to the inflammation of the auditory nerves. This was proved by Dr. Morita's research not to be true. Therefore vitamin $B_1$ is not effective for inflammation of the auditory nerves, but gives vitality to the cells and nerves of auditory organs.

Before discussing prophylaxis, I shall report on related adverse after-effects and their treatment in 39 cases who had been referred to the E. N. T. department from the tuberculosis department as suspected cases of untoward effects of streptomycin; 29 cases were observed for a certain period. One gram of the streptomycin was administered intramuscularly twice a week. In treatment of tuberculosis, when there is an adverse after-effect, the administration of streptomycin should be discontinued and some other antituberculosis drug should be given.

## Treatment

In a case of adverse after-effects of streptomycin treatment, the audiogram shows the lowering of high-pitch sound and gradually that of low-pitch sound. This is well known to all.

If the audiogram shows any change, fortified vitamin $B_1$, 100 mg., is administered intravenously daily. During this treatment audiograms are taken every 10 days. If they show improvement, this treatment should be continued until there is complete recovery. To prevent recurrence the treatment should be continued as follows: the first month twice a week, the second month once a week, the third month two or three

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Department of Otorhinolaryngology, Osaka Communications Hospital.

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times monthly. This treatment should cover a six-month period, and the number of treatments should be gradually reduced. On the other hand, the administration of streptomycin should be discontinued and some other antituberculosis drug given. When the audiogram taken after 10 days shows no improvement or shows progressive adverse effects, the treatment should be continued in order to retard the devolpment of symptoms.

Professor Goto announced after his research that injections—lumbar, carotid, or intravenous—have about the same result in treatment of inner ear disturbances. So the intravenous injection, the easiest of the three, has been chosen.

## Results

Of all the cases examined there were 29 cases that received treatment and had two or more audiograms taken, the first audiogram before the beginning of treatment and the others at intervals of 10 days (during treatment).

In Table 1 the 15 cases in Group I include some who have completely recovered and others with improvement of 20 db. or more with subjective symptoms completely eliminated.

Among this group are included those whose hearing has recovered completely in one ear but is less than normal in the other. Recoveries to normal in audiogram and subjective symptoms at the same time are not infrequent. But these have been included in Group I because their audiograms were not taken before the administration of streptomycin. The fact that though subjective symptoms have not changed, the audiogram shows improvement, must prove that the audiometer is at fault. Unless a method is devised for testing the hearing and differentiating the sound at the same time, a true test cannot be obtained. On the symptom of tinnitus no great research has been done. In the beginning of the appearance of this symptom this treatment will be effective. But even in the early stage recovery may not be possible, depending on where the diseased area is located. For cases of tinnitus which do not recover under the treatment, phenobarbital (Luminal), 0.2 gm. per day can be administered. This will result in lessening and lowering of tinnitus. It has been used in some of the cases.

TABLE 1.—*Results of Treatment*

| Group | Audiogram | Subj. Symptoms | No. of Cases |
|---|---|---|---|
| I | Good | Good | 15 |
| II | Good | Unchanged | 5 |
| III | Unchanged | Good | 1 |
| IV | Unchanged | Unchanged | 8 |
| Total | | | 29 |

## Dosage of Streptomycin and Adverse Effects on Inner Ear

The dosage of streptomycin administered does not seem to be the sole factor in the adverse after-effect. At the time streptomycin was introduced daily injection of 1 gm. for 60 days was considered the maximum dosage. Later when injections of 1 gm. twice a week came to be given, a total of over 60 gm. was used. The symptoms of streptomycin sulfate toxicity appear during the three to six months after treatment. In dihydrostreptomycin injection the symptoms appear during the treatment. The report from the tuberculosis department did not state at first which streptomycin was administered to each patient, but, on further inquiry, it was reported that in the first half of the treatment dihydrostreptomycin was administered and in the latter half combined streptomycin and dithydrostreptomycin.

As shown in Table 2, there were more cases of after-effects among those receiving injections of 30 gm. or more.

TABLE 2.—*Adverse Effects of Streptomycin on the Inner Ear: Relation of Dosage to Number of Cases*

| Dosage, Gm. | No. of Cases | Dosage, Gm. | No. of Cases |
|---|---|---|---|
| 2 | 1 | 31 | 1 |
| 5 | 2 | 39 | 1 |
| 6 | 1 | 40 | 2 |
| 10 | 6 | 42 | 1 |
| 13 | 1 | 45 | 1 |
| 14 | 1 | 50 | 1 |
| 25 | 1 | 60 | 2 |
| 30 | 6 | 80 | 1 |
| | | Total | 29 |

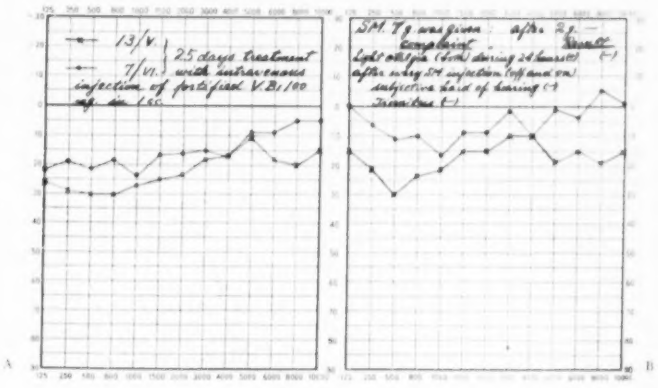


Fig. 1 (Case 1).—Audiogram of 19-year-old woman. *A*, right ear. *B*, left ear.

However, among the cases that received 10 gm. there are six, and even among those injected with 2, 5, or 6 gm. there are some cases. It can be deduced from the above figures that the adverse after-effect depends upon the individual constitution. From the fact that streptomycin does not affect other organs, it must be presumed that it has an affinity for the inner ear. If, when there are adverse after-effects of streptomycin treatment, the symptoms are detected in the early stage and the patient is given prompt treatment, a complete cure is possible. This is a fact known to all, but it is difficult to detect early symptoms. The reason for this is that the adverse after-effect does not depend upon the dosage of streptomycin.

## Notes on Audiograms

Case 1.—A 19-year-old woman showed this audiogram (Fig. 1) after administration of 7 gm. of streptomycin. After administration of 2 gm. she complained of bilateral slight otalgia of 24 hours' duration which started immediately after injection. She did not note subjective difficult hearing or tinnitus. After 25 days' treatment changes in the audiogram were observed and her otalgia was relieved.

Case 2.—A 25-year-old man showed this audiogram (Fig. 2) after administration of 49 gm. of streptomycin. After about 30 gm. he complained of a feeling of obstruction of both ears and tinnitus (Za——) bilaterally. After 63 days' treatment he almost recovered to normal and he has been free from tinnitus.

Case 3.—A 25-year-old woman complained of difficult hearing on the right side and right tinnitus (Pe e e——) following administration of 40 gm. of streptomycin. At present she has received 70 gm. After 35 days' treatment she had almost recovered and she has been free from tinnitus.

Case 4.—A 34-year-old man showed this audiogram (Fig. 4) after administration of 80 gm. of of the streptomycin. He noted a metalic piping sound (Pe——) in the ears in a quiet atmosphere.

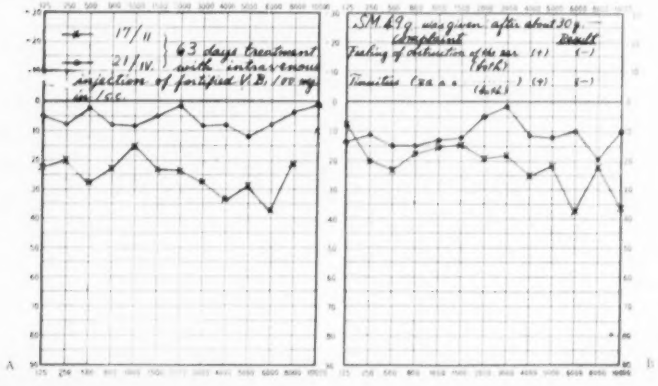


Fig. 2 (Case 2).—Audiogram of 25-year-old man. *A*, right ear. *B*, left ear.

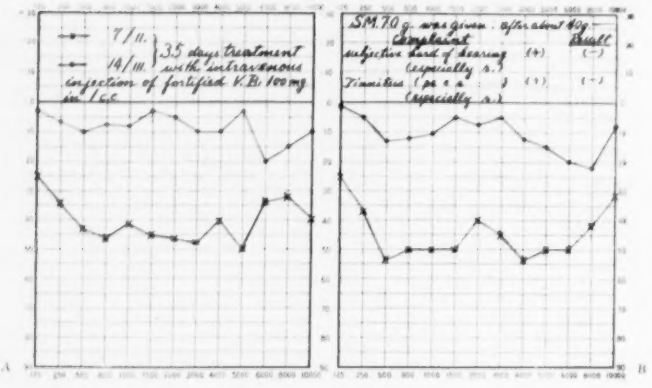


Fig. 3 (Case 3).—Audiogram of 25-year-old woman. *A*, right ear. *B*, left ear.

He noted no difficulty in hearing. After 24 days' treatment he stated that he had been completely cured. But he was short of normality, although his audiogram was somewhat improved.

(Te e e——) occasionally. She denied subjective difficult hearing. After seven days' treatment her symptoms were observed to be progressive. She has not come back.

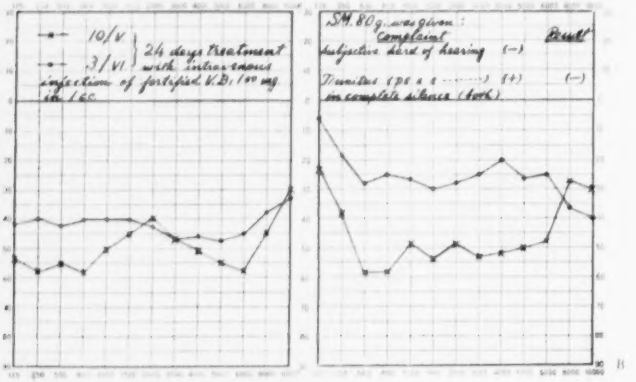


Fig. 4 (Case 4).—Audiogram of 34-year-old man. *A*, right ear. *B*, left ear.

Case 5.—A 28-year-old woman showed this audiogram (Fig. 5) after administration of 42 gm. of streptomycin. She complained of tinnitus

Case 6.—A 41-year-old woman showed this audiogram (Fig. 6) after administration of 67 gm. of streptomycin. After 50 gm. she became

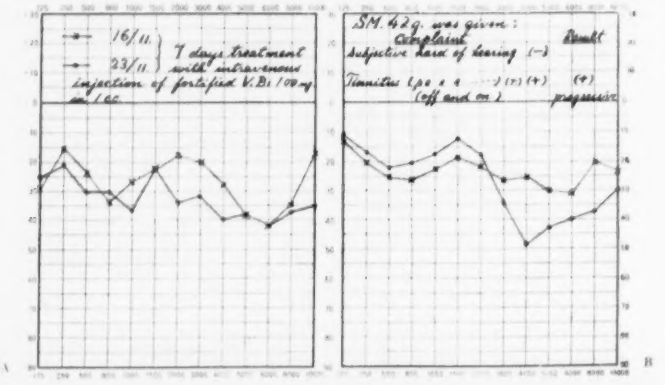


Fig. 5 (Case 5).—Audiogram of 28-year-old woman. *A*, right ear. *B*, left ear.

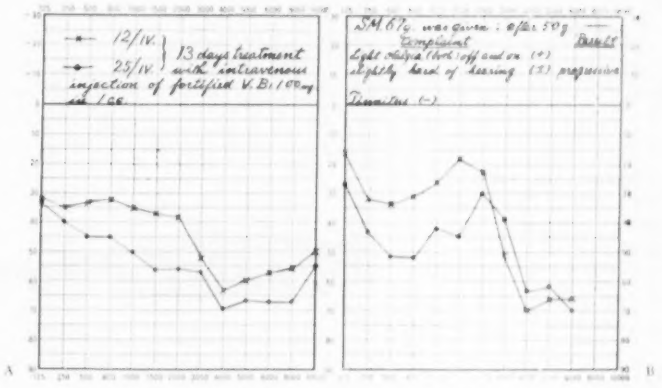


Fig. 6 (Case 6).—Audiogram of 41-year-old woman. *A*, right ear. *B*, left ear.

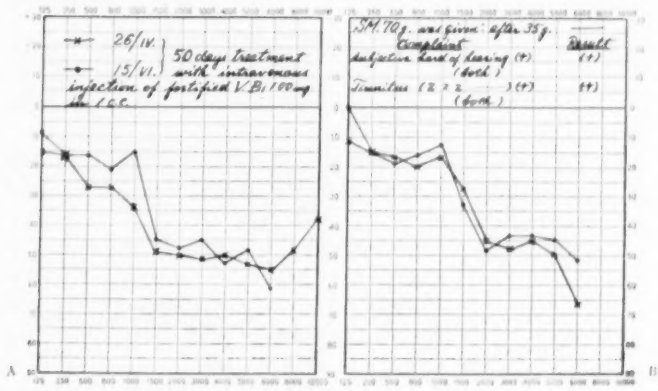


Fig. 7 (Case 7).—Audiogram of 20-year-old man. *A*, right ear. *B*, left ear.

slightly suspicious of difficult hearing in the absence of tinnitus. After 13 days' treatment her suspected symptoms seemed still progressive.

Case 7.—A 20-year-old man showed this audiogram (Fig. 7) after administration of 70 gm. of streptomycin. After 5 gm. he complained of bilateral difficult hearing with a continuous humming sound (Zi——). Fifty days' treatment had no favorable effect.

## Comment

During treatment with streptomycin any adverse after-effect on the ear should be detected as early as possible. The later the detection, the more difficult the cure, even when disturbances on audiogram are comparatively slight. This can be clearly explained by those changes in the ears of human beings and of test animals which are subject to streptomycin toxicity. Here the cells of Corti's organ become confluent, giving an appearance of a single club.

Figure 8 shows the organ of Corti of a 74-year-old woman who was injected with 60 gm. of streptomycin for fatal lung tuberculosis.

The so-called early symptoms are otalgia, feeling of obstruction of the ear, consciousness of compression of the ear, and tinnitus without difficult hearing, which start immediately after administration of the streptomycin and last for 24 to 48 hours. When such symptoms develop, the patient should be tested by audiometer as early as possible in the absence of difficult hearing.

The above-mentioned treatment may afford a favorable chance for the patient. Such a patient is generally found with some form of disturbances in a high-tone scale on audiogram. If he continues to be treated with the streptomycin and little attention is paid to his prodromal symptoms, he will

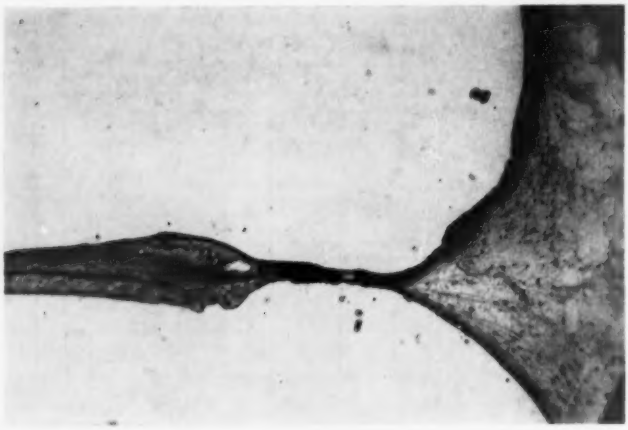

Fig. 8.—The organ of Corti in a 74-year-old woman treated with streptomycin.

develop fixed difficult hearing with a marked impairment in high tones.

In some cases the above-mentioned prodromes are cleared spontaneously in 24 to 48 hours. It is advisable to administer fortified vitamin $B_1$, 100 mg. intravenously, twice, i. e., immediately after administration of the streptomycin as well as the following day, as a favorable prophylaxis against possible adverse after-effects.

Since this method of prophylaxis was introduced, there has been a steady decrease in the number of patients with impaired hearing from the tuberculosis department in the last two years. At present such cases are extremely rare. I have learned from a journal that new drugs similar to streptomycin, without its adverse after-effects have been discovered. But they are said to be in an experimental stage. I hope that the discovery of new drugs can be widely applied in the near future and that the treatment which I have advocated will become obsolete or unnecessary.

## Summary and Conclusions

Twenty-nine pulmonary tuberculosis cases intoxicated with streptomycin (dihydrostreptomycin or combined streptomycin) have been observed.

Ten of these cases, after administration of only 2 to 10 gm. of streptomycin, developed adverse after-effects resulting in impaired hearing.

It is stressed, therefore, that the development of these effects is due not only to amount of dosage but also to individual affinity of the patients.

Fifteen cases were favorably treated with daily intravenous administration of fortified vitamin $B_1$, 100 mg.

Seven cases are presented with their audiograms.

Early detection is possible when impairment of hearing is preceded by abnormal changes in the audiogram.

Early treatment is recommended, because it seems difficult to cure the advanced impairment even though the audiogram shows only slight disturbances.

Early symptoms are slight otalgia, feeling of obstruction of the ear, consciousness of compression of the ear, and tinnitus without difficult hearing, which develop directly after administration of streptomycin and disappear in 24 to 48 hours.

As prophylaxis, intravenous administration of fortified vitamin $B_1$ 100 mg. is suggested directly before or after administration of streptomycin (dihydrostreptomycin or combined streptomycin) with repetition the following day.

Since the introduction of this method of prophylaxis, there has been a steady decrease during the last two years in the number of patients with impaired hearing from the tuberculosis department.

1474 Hatcho Yamato-Takada-Shi.

# The Diagnosis and Treatment of Laryngeal Carcinoma

*A Comparative Study of the Netherlands and the United States*

**SAMUEL B. HENKEN, M.D., Amsterdam**

## Reason for This Paper

If you as a laryngologist were faced with the sudden knowledge that you were a hapless victim of a new growth in the larynx, what type of cure would you select?

The vital question just posed is not new but as old as the knowledge of medical science that cancer is a killer, and that, if not removed or otherwise effectively treated, it may destroy the body with devastating rapidity. Conversely, as a laryngologist, how often is one confronted with this question concerning one's own fate? Most frequently this dilemma applies to a bewildered, mystified, and, maybe, even hysterical (but in any case inexpert) layman patient in whom the specialist has recently diagnosed a new growth.

In spite of the literature that has been published selectively about laryngeal carcinoma during the comparatively brief evolution of its therapy and diagnosis, we must admit that the science of its treatment still is in its infancy. Not more than 30 years have elapsed since radioactive materials were first employed in the battle against it, a little more than 80 years since the first total laryngectomy was done by Billroth, and only a century and a half since Desault first performed a laryngofissure for what he described as "an enlargement of mucosa," which he felt did not belong on the vocal cords.[35] While we bear in mind this short historical résumé, we can safely predict that there is to come a wealth of material to be explored, defined, and added to the scientific knowledge in this field and to make obsolete current findings.

The purpose of this paper is an attempt to define a line somewhere, both in diagnosis and in a convenient form of therapy, based on a simplified and improved nomenclature, so that a more concise, clear-cut, accurate, definite, and unified outline can be established to more clearly set forth the extent and limits of an over-all pattern to insure the most careful and correct removal of a new growth from the standpoint of most effective cure. It is also the purpose of this paper to compare the treatment of laryngeal carcinoma, based on statistical studies and results, in the Netherlands with that in the United States, such studies being based on a comparable era.

As far as these statistics are concerned, they are based on terminology usually employed universally by otolaryngologists in their so-called intrinsic and extrinsic classifications. The "slide rule" employed in transposing these statistics is, simply, that cordal tumors usually comprise those tumors that are confined to the vocal cord per se without any evidence of cordal fixation, and that the so-called extracordal ones include all other laryngeal tumors.[10]

The objective of this simplification is not to arouse arguments as to what the true nomenclature is. No one in his right mind would deliberately stir up a controversy. We all know there is no real winner at the end of a bitter controversy.

This standardization of statistics is an aid in our diligent and logical search for reality. When all authorities approach this

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problem with sincerity and integrity, seeking a solution that is fair to surgeon, radiologist, and patient alike, no controversy will be necessary or wanted. Although there always exists a minority who welcome disputes, we are motivated in seeking a standardized nomenclature to ensure a wiser, more logical, and less distorted diagnosis.

Multiplicity of terminology and statistical standards fosters errors in judgment as a result of confusion. I do not intend to repudiate any present-day accepted opinions. For me to be forward enough to set up these standardized statistics is a bold step, I admit, but it is hoped this paper will be looked upon by otolaryngologists as having as its outcome the inauguration of a universally acceptable guide.

## Evolutionary History of Nomenclature

Since the time that a diagnosis of laryngeal carcinoma was first made, various types of therapy have been advocated by physicians interested in this field. As a result of this, the larynx as a vital organ was studied minutely, anatomically speaking. A direct outgrowth of this study resulted in the division of the larynx into different boundaries, all based, as is known, on certain anatomical landmarks. The otolaryngologists were not the only ones interested in this field, for the radiologists, with the advent of radioactive materials, have long since tried to prove that with their techniques they can dispense with surgery and save the voice box. And now the initial question opening this paper is further resolved: Is surgery alone the ultimate in cure? Or can some form of radiation be employed instead of surgery in order to preserve the speaking voice? It has been suggested that the larynx serves as nothing more than a musical chamber to change the pitch of the voice.[13] Esophageal speech, even though it does not possess the timbre of the speech of the larynx, still can, with some effort on the patient's part, be usefully employed. Such is the argument of the surgeon when he deems a total laryngectomy is indicated. Conversely, the radiologist is not always wrong, for it has been proved that in some cases irradiation is a convenient form of therapy and it can be utilized with the same desirable end-results as surgery, and at the same time preserve the speaking voice.

Laurens[2] at first subdivided laryngeal neoplasms into two groups: One was cancer of the cavity of the larynx, and the other was cancer around its margins. Hautant defined "free cancer of the larynx" as including everything in the area of the larynx and "false cancer of the larynx" as what he found in the pyriform sinus and the postcricoid area. Zuppinger, Schinz, and Coutard[16] defined their terminology of the larynx as (1) laryngeal tumors found in the entire laryngeal area and (2) tumors of the hypopharynx. Krishaber divided laryngeal neoplasms into intrinsic and extrinsic tumors. His intrinsic tumors included those of the subglottic area, the true cords, the ventricle, false cords, anterior and posterior commissures, the epiglottis, and the aryepiglottic folds, while his extrinsic tumors (as he called them) were found in the pyriform sinus and the posterior pharyngeal wall. Hoover and Richter called all neoplasms in the area of the larynx "intrinsic" and everything outside of the larynx "extrinsic." St. Clair Thompson went one step further when he divided laryngeal tumors into (*a*) the intrinsic, which included tumors of the vocal cords per se, the ventricles, the false bands, and the tumors of the posterior commissure; (*b*) the subglottic, which included everything below the true cords; (*c*) the extrinsic, which included the pyriform sinus, the posterior wall, and the laryngeal surface of (1) the arytenoids, (2) the epiglottis, and (3) the aryepiglottic folds. Hayes Martin[31] and Jackson[13] called their classifications intrinsic and extrinsic: The intrinsic pattern included subglottic tumors, tumors of the true cords, the ventricles, and the ventricular surface of the false bands; the extrinsic group included the pyriform sinus, the posterior party wall, the superior surface of the false bands, and the laryngeal surfaces

of (1) the interarytenoid area, (2) the arytenoids, (3) the epiglottis, and (4) the epiglottic folds. Max Cutler's[22] classification was almost identical with that of Hayes Martin and Jackson, except that the former placed the entire false band in his intrinsic grouping.[2] Friedberg's[3] four distinct groupings are (1) carcinoma per se without any evidence of restriction of motility of the true vocal cord; (2) endolaryngeal cancer, i. e., tumor of the vocal cord plus the ventricle and false vocal cords (usually these cases are marked by retarded motility or almost total fixation of the vocal cord); (3) subglottic carcinoma, which may grow either into the vocal cord or down into the trachea; (4) extrinsic or extracordal cancer, including tumors of the area of the epiglottis, the arytenoids, aryepiglottic folds, and the pyriform sinus. Another laryngologist, LeBorgne,[1] of Montevideo, divides laryngeal carcinoma into supraglottic tumors, tumors of the glottic chink per se, and subglottic tumors. He then resubdivides these three groupings into four subgroups: (1) new growth limited to mucous membrane only, with no evidence of impaired motility of the vocal cords; (2) tumors of the vocal cord invading the surrounding area, with psysiological motility of the cord either partially or completely fixed; (3) tumors with their primary origins in the larynx, invading the surrounding lymphatic system with no evidence of palpable neck nodes; (4) primary tumor of the larynx, with definite evidence of neck gland metastasis.

L. H. Clerf's[4] classification nomenclature of carcinoma is cordal, supraglottic, and infraglottic. All others could well be considered as extralaryngeal or extrinsic.

Thomas E. Walsh's[3] classification is the following: 1. Intrinsic tumors are those confined to the true vocal cords when the affected cords are movable. The tumor may cross the anterior commissure and extend down to the opposite cord. 2. Endolaryngeal tumors refer to those found within the voice box, including tumors that involve the ventricle and false cords, with or without fixation of the vocal cord per se. 3. Subglottic tumor is a primary neoplasm that has its origin just beneath the true vocal cord. This tumor usually is extensive when it involves the true vocal cords. A true cord carcinoma that extends supraglottically or subglottically is no longer intrinsic. 4. Extrinsic refers to those primary lesions that involve the epiglottis, the aryepiglottic folds, or the pyriform sinus. Postericoid carcinoma and carcinoma of the party wall are classified as esophageal in origin.

F. C. Ormerod,[35] in his 1953 Semon Lecture delivered in London, gave the following classification: Tumors of the glottis include only those confined to the vocal cord itself. The supraglottic area he divides into the ventricle, the false bands, the middle part of the aryepiglottic folds, and the lower half of the epiglottis. He lays great stress on whether or not the anterior or posterior commissures are involved. He includes these in his intralaryngeal group. His extralaryngeal group contains those tumors that are found on the lateral wall of the aryepiglottic fold and the posterior part of the arytenoid area.

W. H. Struben's[1] classification is as follows: Supraglottic tumors involve the laryngeal surface of the epiglottis and aryepiglottic folds, the false vocal cords, and the ventricles. His glottic tumors are those that arise from the vocal cord per se. The subglottic tumors are tumors originating from the lower edge of the vocal cord down to the inferior margin of the cricoid. These three forms are all classified as intrinsic tumors. His extrinsic grouping includes those that are found on the pharyngeal surface of the epiglottis, the pharyngeal surface of the aryepiglottic fold, the pyriform sinus, and the posterior cricoid area.

Francis L. Lederer,[5] in his textbook entitled "Diseases of the Ear, Nose and Throat," classifies neoplasms of the larynx as (*a*) the intrinsic, those situated on or below the true vocal cords, and (*b*) the extrinsic, neoplasms in the area above the level

of the true cords, including the false vocal cords, the arytenoids, aryepiglottic folds, or pyriform sinuses. He reasons that a simple and brief classification based on anatomic consideration of the larynx (i. e., the true cords "break the lymphatic stream") makes for more ready understanding and easier comprehension.

The above paragraphs present a sampling of the history of the evolution of the anatomical nomenclature of laryngeal carcinoma from a diagnostic standpoint. These classifications were not selected at random, but rather from the viewpoint of showing that otolaryngologists and radiologists concur in their mutual interest in furthering the ever-increasing improvement in the diagnosis of laryngeal carcinoma as an exact scientific guide. This in turn will aid in the over-all international drive for improved cure. The nomenclatures used do not in themselves originate solely in the Netherlands or in the United States but are international in their scope. After all, since when is medicine solely an art and science of one people or one country?

## Present-Day Nomenclatures of Laryngeal Carcinoma

Within the scope of the present-day findings the indications for either irradiation or surgery are based upon a definitely fixed classification of the nomenclature; with this in mind, I present herewith the nomenclatures of the four different laryngeal centers in the Netherlands as contrasted with some centers in the United States. Please forgive me if I do not include all of the nomenclatures in the United States, as I feel that in a great many of them all that is presented is a variation of words, the subject matter being basically the same.

At present in the Netherlands, the scope of treating laryngeal carcinoma usually is directed by the professors in charge of the ENT departments of the four medical university centers, the Netherlands Cancer Institute (the Antoni Leeuwenhoek Cancer Institute of Amsterdam), and the Radio-Therapeutisch Instituut of Rotterdam. It is their classifications that are included in this paper. In contrast, the United States presents a wider variety of nomenclatures that have been evolved by both laryngologists and radiologists. Such being the case, a sampling of American nomenclatures of these two fields will be included.

### A. Definitive Nomenclature in the Netherlands

1. The University of Amsterdam Faculty of Medicine[6-10] and the Netherlands Cancer Institute[6] agree that the grouping includes the boundaries of the inner larynx (endolaryngeal or intralaryngeal). The three classes listed are as follows: 1. Glottic tumors are those of the true vocal cords involving one or both true cords extending to the vocal process or processes. 2. The supraglottic are those tumors that are limited to the mucous lining and are found between the glottis and the pharynx, tumors that extend from the laryngeal ventricle to the aryepiglottic folds, including the false vocal cord. These tumors can also involve the anterior and the posterior commissures and petiolus of the epiglottis. 3. Subglottic tumors are those with or without primary involvement of the true vocal cord.

Their extracordal or extralaryngeal classification includes (1) tumors originating outside of the epiglottis, (2) tumors originating outside of the pyriform sinuses, and (3) tumors originating below the cricoid cartilage.

2. The University of Leiden Faculty of Medicine[7] explains that the intrinsic group includes the subglottic area, the true vocal cords, the anterior and posterior commissures, the ventricle, the false cords, and the arytenoid area.

The extrinsic group includes the aryepiglottic fold, pyriform sinus, base of tongue, and epiglottis. Prof. Dr. H. A. E. van Dishoeck considers neoplasms that originate in the arytenoid area and extend into the posterior party wall as a combination of the intrinsic and extrinsic varieties.

3. At the University of Utrecht Faculty of Medicine [8] the nomenclature of Prof. Dr. A. A. J. van Egmond is outlined in a more detailed manner. His intralaryngeal, or intrinsic, classification is (1) carcinoma of one vocal cord with no impairment of motility, i. e., cancer in situ; (2) tumor of the vocal cord per se with or without impaired motility; (3) tumors originating from the vocal cord and extending to the sinus of Morgagni; (4) tumors involving the entire vocal cord up to, but not including, the vocal process of the arytenoid; (5) tumors originating from the true vocal cord and extending to the false vocal cords; (6) tumors of the anterior or posterior commissure.

His extralaryngeal, or extrinsic, tumors include those of the arytenoid, the epiglottis, the aryepiglottic folds, the pyriform sinus, and those originating from the glottic area and extending subglottically.

4. The University of Groningen Faculty of Medicine otolaryngological service, under the direction of Prof. E. Huizinga,[9] usually employs the following nomenclature: The intrinsic type of noninfiltrative and nondifferentiated forms include (1) cancer in situ of the true cord; (2) cancer involving the entire cord; (3) tumors originating from the true cord and extending to the sinus of Morgagni, with or without involving the anterior commissure; (4) tumors arising from the true cord and extending subglottically, with or without resulting fixation of the true cord; (5) tumors arising from the false band; (6) tumors that arise from the epiglottis, with or without involving the base of the tongue; (7) tumors that arise in the aryepiglottic folds; (8) tumors that arise from the arytenoid but do not infiltrate the party wall. His extrinsic tumors are simply those that arise from the pyriform sinus.

5. The Rotterdamsch Radio-Therapeutisch Instituut [18] divides the nomenclature into these groupings in order to classify tumors according to form of treatment employed: (1) supraglottic, (2) glottic, and (3) subglottic. This simple anatomical subdivision can be used prognostically and therapeutically. Supraglottic tumors have far more metastases than the other two. Uncomplicated glottic tumors have the best prognosis of the three; subglottic, as far as radiation is concerned, offers the poorest prognosis.

### B. Present-Day Nomenclature in the United States

It is not the purpose of this paper to present all the material available in the many laryngeal textbooks published in the United States and available in the many medical libraries throughout the world. Usually the nomenclatures of Clerf,[12] Jackson,[13] Hayes Martin,[31] Lederer,[5] Cutler,[22] Friedberg,[3] T. E. Walsh, and Schall [24] are all employed in their original or variously adapted forms. Most of these have been included under evolutionary nomenclature of laryngeal carcinoma in the preceding section of this paper.

## Definitive Therapy

### A. Surgery

To further comprehend the reasons that a certain treatment is used in removal of laryngeal neoplasms, the following paragraphs will portray, as vividly as possible, the present concept of thinking in regard to surgical removal of laryngeal carcinoma as based on definite anatomical and histological study. Included also in this section is therapy from the radiologist's viewpoint. The reason for the latter inclusion is that, as this is a comprehensive paper, I do not feel I can honestly present one side without also presenting the other's viewpoint, if I am to present a complete and fair report. First will be set forth surgical indications from the ENT departments in the Netherlands' four medical centers, as compared with those used in several of the many clinics in the United States.

I. *Indications for Surgical Intervention in the Netherlands.*—A. At the present time, the Department of Otolaryngology of

the University of Amsterdam,[10] and the Ear, Nose and Throat Service of the Antoni van Leeuwenhoek Cancer Institute[6] employ surgical intervention for removal of laryngeal neoplasms on the following anatomical basis: Laryngofissure is performed in those tumors of the true vocal cord, above and immediately adnexal, which do not cause fixation and which do not reach the arytenoid cartilage.

Hemilaryngectomy is contraindicated at these centers. The rationale presented is that the tumors sometimes treated at other clinics throughout the world by this procedure are classed in the extracordal or extralaryngeal categories with definite cordal involvement. Such being the case, total laryngectomy is a treatment of choice from a standpoint of cure.

The indications for total laryngectomy at both the clinic of the ear, nose and throat service of the University of Amsterdam[6-10] and the Antoni van Leeuwenhoek Cancer Institute are as follows:

1. All cordal tumors that are fixed. (*a*) In all cases of bilateral cordal tumors with fixation of one or both vocal cords. (*b*) Tumors of one vocal cord, with or without involvement of the ventricle and false bands, with fixation of the vocal cord per se. (*c*) Tumors of the anterior commissure with fixation of one or both true vocal cords. (*d*) Tumors of the posterior commissure with fixation of one or both true vocal cords.

2. Those tumors that arise from the laryngeal surface of the aryepiglottic fold or extensive tumors from the laryngeal surface of the epiglottis.

3. Tumors that arise from the pharyngeal surface of the aryepiglottic fold with involvement of the pyriform sinus, with no evidence of the break through of the mucosa.

4. Tumors arising subglottically.

5. Tumors arising from the true vocal cord and extending downward, with fixation of the cord.

B. At the University of Leiden[7] the thinking in regard to surgical intervention, whether or not the tumor be cordal or extracordal, is that surgery is not done as a primary measure. All cases of carcinoma of intrinsic and extrinsic origin are first given a tumorocidal dose of radiation, and, if at the end of six weeks following completion of therapy, repeat biopsy reveals that neoplasm is still present in situ, then surgical intervention is employed. The type of surgery is usually thyrotomy or laryngofissure. Rarely is total laryngectomy employed.

C. Professor van Egmond,[8] at the University of Utrecht, decides the question of surgical intervention on the basis of the following anatomical indications: If the tumor is intralaryngeal, or if it involves the vocal cords, then, according to the anatomical boundaries, the following procedures are employed:

1. In cases where the tumor has originated from the middle third of the true vocal cord and results in partial or complete fixation of the cord, laryngofissure is the procedure of choice. Upon further exploration during surgery, if it is evident that the tumor has involved the cartilage, then fenestration procedure is the operation of choice to complete the cure, if possible.

2. For tumors involving the entire vocal cord up to the vocal process with no evidence of fixation of the cord, laryngofissure is the procedure of choice. In cases where the tumor shows definite fixation of the cord, hemilaryngectomy is the treatment of choice.

3. For tumors that are present on one complete cord and have extended to the anterior portion of the opposite cord, hemilaryngectomy, with removal of a portion of the opposite cord which shows invasion, is the treatment of choice. This procedure is done only where there is no evidence of impaired motility of the vocal cords.

4. In cases where the tumor of one vocal cord has extended to the other one, with fixation of one or both cords, total laryngectomy is the treatment of choice.

5. In regard to leukoplakia or hyperkeratosis affecting the vocal cord or cords, it is

the feeling of Professor van Egmond that these cases are neoplasms of the low-grade malignant variety and not premalignant, as some pathologists believe. Such being the case, he further feels these should be removed by laryngofissure and not treated by radiation, as radiation has not been proved effective.

In reference to the tumors that are classified as intralaryngeal but involve the sinus of Morgagni, the following are the anatomical classifications for elective surgery: 1. For tumors originating from one vocal cord but extending to the sinus of Morgagni on the same side with no evidence of impaired motility of the true cord, hemilaryngectomy is the indicated procedure of choice. 2. In cases where the tumor is felt to originate from the true vocal cord but has extended to the sinus of Morgagni on the same side, resulting in the fixation of the true cord, hemilaryngectomy is still the procedure of choice. 3. In cases of laryngeal cancer of one vocal cord extending to the ventricle and involving the opposite cord with total fixation of, or partial impairment of, the original vocal cord, total laryngectomy is the procedure of choice. 4. For tumors originating from the true cord and extending to the false cord with fixation of the same true cord, hemilaryngectomy is the procedure of choice. However, total laryngectomy is done if the tumor has spread to the opposite true cord. For tumors originating from the anterior commissure with no evidence of cordal involvement, laryngofissure is the procedure of choice, and with this procedure only the involved portion is removed.

Total laryngectomy is done when tumors of the larynx are found to involve the posterior commissure with no evidence of vocal cord fixation.

At the laryngology service at Utrecht, extralaryngeal, or extrinsic, tumors are treated by total laryngectomy, and the following conditions must first be satisfied before this procedure is performed: 1. For simple tumors involving the epiglottis, epiglotidectomy is the procedure of choice. (However, if the neoplasm is extensive, total laryngectomy is performed.) 2. For primary tumors of the aryepiglottic folds, total laryngectomy is the procedure of choice. 3. Tumors of the pyriform sinus. 4. Tumors originating from the true vocal cord and extending subglottically with or without fixation of the true cord.

Radical neck dissection is not frowned upon at this clinic. However, when it is performed in conjunction with total laryngectomy, it is usually followed through with irradiation. The neck dissection is done whether cervical nodes are or are not palpable.

At Utrecht surgery is the treatment of choice in all cases where primary laryngeal neoplasms are proved to exist, except in the following three cases: (1) cancer in situ and proved cancer of the middle third of the vocal cord with no fixation (in these cases radiation therapy is the treatment of choice), (2) when surgery is deemed inadvisable because of social conditions of the patient, i. e., when the voice is essential for employment, or (3) when surgery is refused.

D. At the University of Groningen Faculty of Medicine,[9] definite surgical intervention methods are either laryngofissure, hemilaryngectomy, or total laryngectomy. The indications for laryngofissure are limited to tumors of the true vocal cord with no evidence of cordal fixation. If, upon examination, it is found that the tumor has spread to the anterior commissure, this type of procedure is not contraindicated. It must be borne in mind that the tumors are histologically proved nondifferentiated.

Since 1951 all small tumors, limited to one freely movable vocal cord, have been treated with x-ray, up to now with good results. Laryngofissure is performed instead, if individual circumstances make an unimpaired speech organ less essential.

Hemilaryngectomy is employed in cases where the tumor arises from the true vocal

cord and extends to the thyroid cartilage with or without evidence of cordal fixation.

Total laryngectomy is done in all other cases of intrinsic or of extrinsic origin, depending on whether or not this procedure can be utilized as a life-saving measure and if the age, physical condition of the patient, or extensiveness of the tumor does not preclude it.

II. *Indications for Surgical Intervention in the United States.*—According to Louis H. Clerf,[4] the indications for surgical treatment of laryngeal carcinoma are as follows: "A small discrete superficial carcinoma of a vocal cord can be treated by peroral methods. It is imperative that this be a very small lesion often described as carcinoma in situ."

Laryngofissure should be limited to a discrete carcinoma of one vocal cord. This must be freely movable; partial laryngectomy is done in cases of carcinoma of a vocal cord which has crossed the midline anteriorly but in which there is no fixation of the vocal cord or subglottic extension.

Total laryngectomy is performed on all lesions of a vocal cord with fixation of the cord or of an arytenoid, where there is subglottic extension, and in all supraglottic lesions. If the lesion is extensive, an elective block dissection on the same side of the neck should be done en bloc at the same time as the laryngectomy. If lymph nodes are palpable but not fixed, a combination total laryngectomy with en bloc dissection of the neck should be performed.

C. L. Jackson,[11] in a recently published article stated that his two types of laryngofissure are to be used under certain specific conditions. His first type is the "clipped procedure," and he utilizes this for lesions of the middle third of one cord with good motility. His other version, known as the "anterior commissure procedure," is employed in cases where the lesion involves or approaches the anterior commissure. Total laryngectomy is done in cases where the tumor is cordal but by direct extension has produced impairment of motility or has extended supraglottically or subglottically. The lesions included in this group can extend to the false cords and/or ventricles and also invade the cartilage. A surgical procedure, not done for the last past 20 years in the Netherlands but employed in some clinics in the United States, is the laryngeal suspension procedure. Indications for this type of procedure are (1) early cordal lesions, (2) cordal lesions, and (3) cordal lesions with fixation or extension.[23]

Surgical opinion varies to a slight degree, but the general salient indications for surgical intervention are usually based on the following pattern. When laryngofissure is to be employed, the cases to be selected are (1) tumors that involve the vocal cord up to the vocal process posteriorly and the anterior commissure anteriorly (without evidence of cordal fixation); (2) tumors of the vocal cord, including the anterior commissure, with no cord fixation; (3) tumors that are cordal in origin and have reached the vocal process with some evidence of retarded motility of the vocal cord; (4) those tumors that are confined to the vocal cord; (5) tumors that are confined to the middle third of the true cord and by pathological study have been found to be noninfiltrative, i. e., cancer in situ. In lesions involving the anterior commissure with no cordal fixation, it is the opinion of Clerf [12] that these usually follow laryngofissure, and he advises laryngectomy as the procedure of choice. In some clinics hemilaryngectomy is done in certain selected cases of laryngeal carcinoma. Usually in these cases the tumor arises from the true cord and extends to the sinus of Morgagni and false bands with or without evidence of vocal fixation. Total laryngectomy [13] is in the main employed when there is definite fixation of some degree of the involved true vocal cord and when the tumor has its point of origin on the same cord. The following is a résumé of the indications for this type of procedure: (1) tumors of the vocal cord that have extended to and included the vocal process and at the same time have produced

impairment of motility of the vocal cord; (2) tumors of the vocal cord that have extended subglottically and at the same time have produced impairment of the vocal cord; (3) tumors that have originated from the vocal cord per se and have extended into the ventricle with impairment of the vocal cord; (4) tumors that have originated in the vocal cord and by continuity have spread into the ventricles and false bands, resulting in impairment of motility of the true cord; (5) those tumors that have originated in some point in the larynx and have invaded the cartilage with impairment of motility of the vocal cord per se. The fifth group can be further subdivided into the following: (*a*) tumors of the epiglottis (laryngeal surface) that are found near the petiolus, resulting in fixation of one or both vocal cords; (*b*) those tumors arising from the aryepiglottic folds (laryngeal surface) which have extended into the arytenoid cartilage, resulting in impairment of function of the vocal cord; (*c*) tumors having their origin in the posterior commissure, arytenoid area, with the fixation of the vocal cord, one or both vocal cords, without any evidence of infiltration of the party wall (these tumors are thought by some authors to be esophageal in origin and as such are contraindicated for this type of procedure); (*d*) subglottic tumors that have extended into the larynx and have resulted in partial or complete fixation of one or both true vocal cords; (*e*) tumors originating in the preepiglottic space (regarding these, it is thought that because of the relative frequency of cervical node metastasis a radical neck dissection should be performed in conjunction with a total laryngectomy). In the cases of intrinsic lesions that originate from the larynx but invade the pharynx or the base of the tongue, the laryngopharyngectomy procedure as is described by Hayes Martin [32] or the lateral transthyroid procedure of H. B. Orton [29] offers a good chance for cure.

It is the recent consensus in the United States that a total laryngectomy when used as a cure for the so-called "extrinsic" carcinomas of the larynx, especially if it is widespread, in itself will not remove the neoplastic growth. It has been definitely shown that, even though the primary neoplasm was removed, in 39% of all cervical nodes examined tumor was found to be present.[14] It can be broken down further into about 40% cancer infiltration into the cervical lymphatics in laryngeal neoplasms whose origin is supraglottic and about 17% when they arise infraglottically.[15] In brief, several laryngologists in the absence of palpable cervical nodes advocate prophylatic neck dissection en bloc with total laryngectomy for certain specific conditions [3,12,14]—(1) tumors orignating from the preepiglottic space, (2) extensive supraglottic tumors with definite cordal fixation, and (3) tumors originating subglottically with definite cordal fixation. In the conditions listed, this type of procedure can be regarded as the only hope for complete cure and removal of the disease.

### B. Radiation

As we have seen, surgical intervention plays a paramount role in treatment for carcinoma of the larynx, especially so when it is based upon sound medical practice. Likewise, ever since the introduction by Coutard [16] of radioactive substances in his management and therapy of laryngeal carcinoma, radiation has been definitely proved by statistics of results to have its place in modern laryngeal carcinoma therapy. The question of its value in this role is not debatable, for in some instances the results are as good as those obtained by surgical intervention; and at the same time the speaking voice is preserved. However, we must remember to bear in mind that, with the patient's exposure to the rays, a possible perichondritis, dysphagia, and, to a less extent, replacement fibrosis of the surrounding healthy tissues may result. These manifestations, even though they do not occur in all cases where the patient has been exposed to radiation, nonetheless do occur. Whether this condition is a direct result of improper

use of the radioactive materials or an inherent manifestation of the patient I cannot answer. Meijer,[2] in a follow-up of the many cases of irradiated carcinoma patients at the Antoni van Leeuwenhoek Cancer Institute, has proved that the appearance of perichondritis is almost the rule when the irradiated cartilage is diseased beforehand, especially when the growth of carcinoma into the cartilage has taken place. This is the reason that in Amsterdam[6] patients with immobilized cords are never irradiated, because an immobile cord proves the existence of tumor cells outside of the mucosa and therefore probably also in the perichondrium and the cartilage. This is one author's opinion.[2] However, there is always hope that improving the technique will modify or obliterate the resulting tissue destruction. This same clinical picture is present not only in the Netherlands but also in the United States.[33]

*1. Definitive Indications for Radiation in the Netherlands.*—In the Netherlands current indications for radiation intervention in some form are based on anatomical divisions. At the department of otolaryngology[10] of the University of Amsterdam and at the Cancer Institute in Amsterdam,[6] the classification employed is as follows: 1. Supraglottic tumors that take their origin from the ventricle or from the false vocal cord which, because of their gross appearance, present a superficial necrosis. These tumors are not amenable to minor surgical intervention. 2. Tumors that arise from the true vocal cord and that extend to the supraglottic area, especially those tumors that show an undifferentiated cell structure histologically. These cases are indicative, provided there is no fixation of the cord. 3. Extralaryngeal tumors arising from the pharyngeal surface of the aryepiglottic fold, pyriform sinus, and the pharyngeal surface of the epiglottis.

At the University of Leiden,[7] as has been already mentioned, tumors of the larynx are treated initially by some form of irradiation, and I do not believe that anything else could be added at this time.

Professor van Egmond's[8] clinic at Utrecht uses radiation therapy when the following conditions are met: (1) cancer in situ; (2) when surgery is deemed unnecessary because of the social handicap that it may cause, such as employment difficulties (this therapy is used only when the tumor has involved only one vocal cord and there is no evidence of fixation); (3) where surgery is declined; (4) where surgery can not be done because of the presence of chronic illness, advanced age, or when the physical impairment of the patient contraindicates surgery.

Radiation therapy at the clinic of the University of Groningen[9] is restricted to the following conditions: (1) intrinsic tumors localized to the true vocal cord itself with no evidence of cordal fixation and no evidence of infiltration into the anterior or posterior commissures and (2) extrinsic tumors, i. e., only those whose origin is from the pyriform fossa and the laryngopharynx and are so extensive that x-ray therapy as a palliative measure alone can be used.

It is the opinion of Professor Huizinga[9] that in the cases described under the heading of intrinsic tumors radiation therapy be used only when the age group in which the patient falls and economic circumstances preclude surgical intervention, i. e., when the age of the patient makes it necessary for him to earn his living by the use of his normal voice, and when therefore loss of the voice would be a hindrance to him.[10]

Dr. K. Breur,[18] of the Rotterdamsch-Therapeutisch Instituut, bases his indications for radiation therapy on whether the tumors are what he calls supraglottic, glottic, or subglottic.

1. Supraglottic tumors: In general, radiation therapy is used. Surgery is used when extensive involvement of the cartilage is evident. In each individual case the spread of the tumor, the way of infiltration, the histological type of tumor, the general condition of the patient, and the social status have to be taken into account before decid-

ing means of therapy. After radiation a close follow-up is advised, and surgical therapy is recommended at the first sign of recurrence.

2. Glottic tumors: A. Cancer in situ cases, in his opinion, should be "observed" only if good follow-up is possible. Inspection of the area should take place every two months during the first year. If there are signs of further growth, another biopsy is indicated. From the point of view of radiobiology one cannot expect these cancers in situ to be radiosensitive, as they are extremely well-differentiated histologically. A conservative approach is advised because (*a*) surgical removal will lead to impairment of the voice and (*b*) there will not be any chance to prove that these histological pictures will give rise to actual tumor growth if the cord is taken away or heavily irradiated. Only if it can be proved in the future that a great percentage of these conditions will lead to cancer, is a more active attitude justified. B. Leukoplakia, hyperkeratosis, and papillomata can be considered as premalignant and radioresistant; surgery is advised. In a few cases of recurrent papillomata radiation therapy can be of some help. C. Extremely well defined carcinomas of the vocal cord, i. e., those arising from leukoplakia, are cases where surgical removal of the cord is advised. In selected cases of small sharply defined superficial tumors roentgen contact therapy during laryngofissure can give fine functional results. D. In less differentiated tumors, the therapy consists of radiation from two lateral portals, with careful follow-up. On the first sign of recurrence, laryngectomy as soon as possible is indicated.

3. Subglottic tumors: Preference is surgical therapy. Tumors of the pyriform sinus invariably come under treatment with extensive lymph node metastases, these metastases being in many cases the only symptom. In these cases prognosis is very poor, and in Breur's opinion these tumors should be classified in his hypopharyngeal group.

*II. Indications for Radiation Therapy in the United States.*—In contrast to the above-mentioned indications for the employment of radioactive materials as used in the Netherlands, somewhat different indications for their use have been proposed by varying authorities and are employed by clinics in the United States. (Not all of the proposed nomenclatures necessarily are in actual use.) Herewith is presented some of the American nomenclatures. According to Garland's [19] classification and "staging," cancer of the larynx is an intrinsic tumor that arises from the true cord or laryngeal ventricle. These cancers are divided into three stages: stage one—single cord without fixation; stage two—both cords, with cartilage invasion or metastasis. And extrinsic tumor arises from the upper surface of the false cord, epiglottis, aryepiglottic fold, arytenoid, laryngeal surface of pyriform sinus, or postcricoid area. These stages are further subdivided into extent of lesion, and he describes them as follows: stage one—less than 1 cm. in diameter; stage two—from 1 to 3 cm., but without cartilage invasion. Stage three—more than 3 cm., or with cartilage invasion or metastasis.

A composite picture of indications employed by Maurice Lenz [20] is as follows: X-ray therapy can be employed in all carcinomas of the vocal cord except when the cancer has extended into the arytenoid cartilage or has extended subglottically. He further feels that radiation therapy in early cancer of the vocal cords gives as high a cure rate and retention of the speaking voice as does surgical intervention. However, it is this radiologist's opinion that surgical intervention is the procedure of choice, followed by irradiation in all other cases where feasibility exists.

J. A. del Regato [21] feels that radiotherapy achieves its more constant and best results in the treatment of early noninfiltrating carcinomas of the vocal cord. He further feels that results of radiotherapy when employed in combating carcinomas of the laryngeal vestibules will compete favorably with those

of total laryngectomy for these same groups of tumors. However, he does not feel that radiotherapy would be of much value primarily when there exists carcinoma of the retrocricoid and subglottic regions or of infiltrating carcinoma of the vocal cord per se. As a radiologist he feels that surgical intervention is the treatment of choice if practicable, especially when cervical node metastasis exists.

Max Cutler's[22] criteria for selecting laryngeal carcinoma for radiation treatment are as follows: 1. General—age and general condition, life expectancy, and presence or absence of organic disease. 2. Extent of lesion. If very advanced, pallative irradiation only is given. 3. Site of origin. Lesions arising in the ventricle, false cords, or base of epiglottis are radiosensitive. Sensitivity of tumors of the true cords depends upon extent and degree of infiltration. 4. Microscopic structure. The prognosis cannot be predicted except that anaplastic malignancies have a bad outlook. 5. Mobility of laryngeal structures is of good prognostic value, except in cases of fixation, which is often due to inflammation, especially after biopsy. (The value of this last mentioned factor has been questioned by some authorities.)

However, it is my opinion that in Cutler's mode of selection of cases not enough care was exercised in defining the limits of anatomical extent, and I believe this results in too wide a latitude for error when considered from the viewpoint of this study.

To say that the above descriptive paragraphs relating to definitive radiological and/or surgical therapy in the Netherlands and in the United States is complete would be a misnomer. I do not intend to condense all previously published textbooks and articles or to replace all professors and technicians in these fields. It is my intention to present only a contrasting picture in order to ascertain definite criteria for selection of therapy. This is made a little difficult by the fact that radiologists have published less material to explain their viewpoint than have surgeons. In this paper we are looking for quality, as well as quantity. I hope that this paper will not be looked upon with bias but that it will be accepted with an open mind to promote improved therapy.

## Statistics

### A. Netherlands

The proof of the effectiveness of the method of treatment lies in the results. To this end a comparative study was made of statistics compiled both in the United States and in the Netherlands in a comparable era. However, it must not be lost sight of that during World War II the Netherlands was isolated from outside medical influence and this period included a part of the era covered by this study. As the result of this retarding condition the following compiled statistics may not be an exact index from a purely scientific viewpoint.

Regarding statistics in the Netherlands, the data will be divided into five groups to represent the four different medical centers and the Rotterdamsch Radio-Therapeutisch Instituut.

J. S. J. Meijer,[2] reporting on 224 cases of laryngeal carcinoma during a period from 1923 to 1948, presented the following statistics. These cases were seen and treated at the Antoni van Leeuwenhoek Cancer Institute in Amsterdam. These 224 cases were divided into four therapeutic divisions: 1 (*a*) Nineteen cases were included in this division; each case presented cancer of the true vocal cord with definite evidence of cordal fixation. The mode of therapy was hemilaryngectomy followed by low doses of radiation. Of these cases 3 remained alive and well after five years, 6 died within five years from causes other than the primary disease, while the other 10 succumbed as a direct result of it. The percentage of cures in this study group was 16%. (*b*) Fifty-four cases were included in this grouping. All presented true cordal tumors with no evidence of cordal fixation. Of this group 41 remained alive and well after five or more years; 6 died from causes other than the primary disease, while 7 died

from recurrences of the disease within five years. Total percentage of cures was 76%. The mode of therapy was laryngofissure. 2. In this grouping were 79 cases of true cordal tumors with no evidence of cordal fixation. The mode of therapy was radiation according to the method of Coutard.[16] Twenty patients were alive and well after five years, while the remainder died from intercurrent infection, recurrence of the primary disease, or as a direct consequence of radiation. Total percentage of cure was 25%. 3. This division was comprised of those cases of extracordal carcinoma with definite evidence of infiltration and vocal cord fixation. The mode of therapy was hemilaryngectomy followed by radiation. Out of a total of 16 treated cases, 5 remained well and free from disease five or more years. The others succumbed to recurrences within the five-year period. Total percentage of cures was 31%. 4. These cases presented definite evidence of true extracordal tumors. The method of therapy was by total laryngectomy. Of 21 cases only 4 remained alive and well after five or more years. Of the other 17 cases, 11 succumbed from recurrence of the primary disease, 4 died as a result of surgery, while 2 died of intercurrent infection. The total percentage of cures was 19%. The remaining cases not included in the above groupings were those cases that were not definitely included in the ear, nose, and throat service. For statistical study they were not included in this survey. It must be further borne in mind that the selection of therapy used in this study of Meijer's [2] did not follow the pattern of nomenclature that is employed today in Amsterdam.[6-10]

Statistics compiled from Utrecht [8] during the period of 1939 to 1949 with a review of 30 cases of laryngeal carcinoma revealed the following results: In the intrinsic category there were five cases and in each of these a laryngofissure was done. The five-year cure rate was 40%. When radiation was employed in four other cases, two remained well after five years, for a 50% cure rate. In the extrinsic grouping there were 19 cases. All had total laryngectomy. Of these 19 cases, 12 remained well after five years. This was a 63% cure rate. A combined laryngopharyngectomy was done in two cases; both of these cases were living and well after five years.

Out of the total of 35 cases reviewed from 1939 to 1949 at the laryngological service at Groningen,[9] the statistics can be broken down into those that had undergone laryngofissure and hemilaryngectomy, as the indications are almost alike. There were a total of 18 cases in this group. Ten of these cases were known to be alive after five or more years. The percentage of five-year cure was 54%. In cases of extracordal carcinoma where a total laryngectomy was performed, out of the total of 17 cases, 8 were alive and free from disease five years or more. The percentage of living is 47%. Eight cases received radiation therapy for extrinsic carcinoma. Of these eight there were no five-year cures, death having occurred anywhere between one and five years, presumably from this disease.

Statistics compiled from the Rotterdamsch Radio-Therapeutisch Instituut [18] during the years 1945 through 1951 show that out of a total number of 134 cases reviewed, 55 of these patients were in the supraglottic grouping; 13 remained alive and well and free from disease three or more years. The total percentage of cure was 24%. Seventy-six cases received treatment for carcinoma of the true glottic grouping. Of this group 37 remained alive and well and free from disease three or more years. A 49% cure rate was obtained from this group. Of the three cases treated for subglottic carcinoma, only one remained alive and well and free from disease three or more years. Obvious percentage of cure is 33%.

The University of Leiden [7] failed to supply statistical data.

### B. The United States

Herewith are presented some statistics from the United States. These statistics, as

has been stated previously, were those selected from a comparative era. Not all are listed, as I feel that a great many of them are repetitive and repetition does not add to the clarification of the results. The tabulations, in so far as surgery and radiation are concerned, are listed to insure an over-all picture from both therapeutic branches. Furthermore, all parts of the country are represented in this statistical survey, if geographical distribution has any significance concerning end-results.

From the standpoint of surgical intervention in the cure rate of laryngeal carcinoma, the following results were tabulated: Chevalier L. Jackson,[11] reporting on a series of 181 cases of so-called "intrinsic" carcinoma via laryngofissure from 1930 to 1947, concluded that of these cases 158 remained free from disease after five or more years, a percentage of 87%. Recurrence or metastasis resulted in 20 other deaths, while the rest were due to die from either operative mortality, metastasis to other parts of the body, or other causes.

F. E. Lejeune,[23] reporting on 59 cases via the transoral route, recorded 41 patients who survived five or more years. Fifteen other patients were reported as free from the disease from one to five years. The remaining three have had recurrence, necessitating laryngofissure for removal of the new growth, and are reported as being well at this time. Total percentage of cures via the initial procedure was 70%.

LeRoy A. Schall,[24] reporting on 165 patients with cancer of the larynx who were treated by total laryngectomy for extracordal or extrinsic cancer, reported 100 patients, or 61%, were alive and well five or more years.

J. J. O'Keefe,[25] reporting on 193 patients with cancer of the larynx in whom laryngofissure was performed, noted that 117, or 61%, of the cases were free from the disease five or more years. The indications for this procedure were only those tumors that were restricted to the anterior portion of the middle third of the vocal cord without involvement of the anterior commissure or extension to the opposite cord. It is interesting to note that of the 176 patients treated by laryngectomy for extrinsic laryngeal neoplasms, only 81 persons, or 46%, were alive five or more years, and in this group 56 cases of recurrences were noted, 41 to the cervical lymphatics and 16 to other organs.

Kirchner and Malkin[26] recorded statistics compiled from a total of 235 cases of proved cancer of the larynx where laryngofissure was thought indicated and done; there was a 67% five-year or more cure rate.

McCall and Fisher,[27] reporting on 104 cases of cancer of the larynx, compiled the following statistics: Of those patients that were diagnosed as having "intrinsic" carcinoma and treated by laryngectomy, 90% were alive and well five or more years, while only 21% who had undergone total laryngectomy for extrinsic carcinoma were alive and well five or more years.

In contrast to the surgical results, the compiled statistics taken from radiological services throughout the country reveal that in certain instances their results were as good as those obtained through surgical intervention.

L. H. Garland and M. A. Sisson,[19] report the end-results in 62 cases of primary cancer of the larynx from the period 1933 to 1946. The classification that is employed in this grouping is not what the laryngologist usually calls true cordal neoplasms, for in their first stage, tumors are included where there was definite evidence of cord fixation and with it infiltration into the surrounding lymphatics. However, in a review of the three classifications, in their so-called "intrinsic" group, the first division only contains those cases where there is no cordal fixation, and of the two reported cases, both remained alive and well five or more years. In the remainder of their reported cases, including those of their extracordal grouping (and these can all as well be grouped under the heading of extracordal neoplasms) only 26% of the cases remained

alive and well and free from the disease five or more years.

Maurice Lenz[20] reporting on a group of 110 cases of laryngeal carcinoma and using his classification, i. e., including those that we know as extracordal, compiled the following statistics. Of the well-differentiated carcinomas of the vocal cord, 27% of the total remained free from disease five or more years; 26% of the moderately well-differentiated carcinomas remained free after five years, and 37% of the undifferentiated tumors remained free from the disease five or more years.

LeRoy A. Schall,[28] reporting on a series of patients who had undergone external irradiation for extracordal carcinoma and external irradiation for cancer of the vocal cords, noted that of the former he obtained a cure rate of 18%. However, in regard to the latter category, he obtained a cure rate of 92%. It must be borne in mind that the laryngologist reporting on this group does employ both surgery and radioactive therapy when treating laryngeal carcinoma.

I hope that the above-mentioned statistics will not be looked upon as a complete review of both types of therapy, but rather as a comparative study to illustrate the pitfalls in basing end-results on nomenclatures that are not universal. I have tried to revise the nomenclatures so that any given indication for therapy and the statistics compiled pertaining thereto are as closely paralleled as possible in order to facilitate the presentation of a comprehensive contrasting picture.

## Summary

A comparative study of the nomenclatures of laryngeal carcinoma in the United States and in the Netherlands reveals that at this time they are basically about the same. Therapeutically speaking, if it were not for the fact that the well-differentiated nomenclatures could be more standardized, the indications for selective treatment for the most part are the same.[6]

A review of the statistics reveals that there is a higher cure rate where the indications are for laryngofissure in the United States as compared with the Netherlands in a comparable era. However, I do not lose sight of the fact that the Netherlands was an isolated country during the Second World War, and, medically speaking, we were not able to profit by an interchange of ideas with other countries. This last factor can also be applicable to indications for laryngofissure. At this time, however, a short time review of the present-day results leads me to feel that the cure rate in the Netherlands will compare favorably with that in the United States when statistics are compiled on a current five-year or more basis.[6,34]

Statistics on total laryngectomies reveal a well-differentiated pattern, and again we find the factor of the Netherlands' isolation during World War II and the very poor follow-up for patients after surgery during that period. I make this statement based on statistical study from a comparative era.[2] It appears evident that the latent conservativeness of the operating surgeons was not justifiable, when the indications for total removal of the larynx were concerned, in so far as comparative studies of the two countries reveal. Whether or not this can be due to indications based on improper nomenclature or hesitation about removal of a vital organ is a question I cannot truthfully answer.

Radiation, when employed for true cordal tumors, shows about the same cure rate, comparatively speaking. However, when the tumor has spread and become infiltrative, the quoted statistics reveal a wide discrepancy. Whether we can attribute this to finer diagnosis or better technique is a moot question. A review of the comparative study further reveals that radiation, no matter how good the technique, will not give the same results for extracordal tumors that surgery will give. It appears evident that radiation can arrest but will not cure infiltrative tumors.

In the evolution of the nomenclatures of laryngeal carcinoma up to this present writ-

ing and with it a fuller comprehension of diagnosis and treatment, it appears very striking that the nomenclature can be so simplified that treatment can be made dependent upon it. In short, it appears evident that in cases where the tumor is confined to the true vocal cord, or in its immediate supraglottic area with no evidence of infiltration, a laryngofissure, the transoral suspension procedure, or radiation can be very well utilized. However, the vocal process must not be involved. It can be proved anatomically that, such being the case, either one of the described treatments can justifiably be utilized, provided, of course, that the tumor is one that is nondifferentiated from a histological viewpoint. Furthermore, because of the rich lymphatics present above the cord, too much spread of the cordal tumor could not be included in this grouping. Such being the case, the supraglottic limits could include only the cordal end of the ventricle. These have been referred to in the past as intrinsic tumors, endolaryngeal tumors, or true cordal tumors. Apparently they all have reference to one and the same thing. For brevity's sake I would suggest that these be called cordal tumors.[10]

Once the tumor has become infiltrative and has spread beyond the confines of the true cord, even though the function of the cord may not be impaired, we could not call this a cordal tumor because of its infiltrative nature. And because of the anatomical proximity of the regional lymphatics, the supraglottic tumors are more infiltrative than the infraglottic ones. Up to this present writing, laryngofissure has been utilized in certain of these conditions where the tumor has spread to the inferior border of the false cord, or when the tumor has infiltrated into the anterior or posterior commissures. Knowing that the lymphatics spread the disease once the tumor has become infiltrative, could we honestly classify these tumors as cordal? Hence, should not these tumors be called extracordal?[10] From an anatomical point of view the chances of cure are with laryngectomy and not with partial removal of the larynx. To say that this point is controversial is wrong. I base this point on statistical studies.[2,8,9,11,18,20,23-28] What other basis can we use?

Regarding the other types of laryngeal neoplasms, they are all called extrinsic or extracordal. Could not these be included within the catagory of extracordal tumors, as total extirpation of the larynx is the only procedure that we could employ for a safeguard measure of cure? By this statement I mean only those tumors whose primary site of origin is not the true vocal cord. We know from anatomical studies that there is no paucity of lymph nodes in the area, and by the time the patient is seen the biopsy reveals a markedly infiltrative tumor. Within the same catagory of extracordal tumors, we can place those tumors that are found in the pyriform sinus. These tumors cannot be called true laryngeal tumors, but by the time the patient is seen it is a moot question whether the point of origin is from the pharyngeal surface of the aryepiglottic fold or the arytenoid or from the laryngeal surface. We have seen that these tumors are just as amenable to surgery and have a better cure rate than with radiation; and the recent article of Henry B. Orton,[29] of Newark, N. J., will bear out this factor.

Little or nothing has been said or expressed in past nomenclatures regarding pachyderma, keratosis, and papillomata of the vocal cords,[6] with the exception that, pathologically speaking, these tumors are usually premalignant. If these are confined to the true vocal cords and, as such, are true cordal tumors, they should be included in this catagory. The word "premalignant" in itself should convey a definite message to us. Knowing that once it becomes malignant it can kill, should we wait until it becomes that? Or, should we remove it by surgery before it has a chance to become malignant? This question up to the present time is controversal; several writers and teachers[3,6,9] treat this condition by various methods, from repeated biopsy to irradiation. In the Netherlands treatment for this condition

ranges from surgery to repeated biopsy and observation. Radiation so far has not definitely been effective. The only other remaining therapy for cure is laryngofissure. This method of therapy has been utilized by W. H. Struben [1] in certain cases of leukoplakia and hyperteratosis of the vocal cord with favorable results in the past few years. In rebuttal some may say that this approach is a bit too radical, as this type of tumor usually is found in the older age groups when neoplastic growth is slow and the disease is limited to the true vocal cord. Whether or not it can be placed in the infiltrative type group is not too well known; but it does not appear that radiation can guarantee us permanent cure, which I hope is what we are looking for.

Prophylactic neck dissection is not a new surgical approach in therapy for laryngeal tumors as it was considered several years ago. Sorenson and Gluck in their writings will attest to this. History reveals that since then Hayes Martin,[31] Clerf,[12] Ogura and Bello [14] and more recently H. B. Orton [29] have advocated radical neck dissection with total removal of the larynx in cases where the neck nodes are not palpable. In a recently published article, this opinion and this type of procedure were shown to be a safeway measure, especially in those tumors that were subglottic, in massive epiglottic lesions and others in this group. H. B. Orton [29] advocates a bilateral neck dissection in the absence of palpable neck nodes in cases where the primary tumor is found to be in the pyriform sinus, tumors that extend from preepiglottic space to the base of the tongue, and tumors whose origins are from the arytenoid area and extend into the pyriform sinus. Prophylactic neck dissection has not as yet been looked upon favorably in the Netherlands. It appears to be the consensus that this type of procedure is rather unnecessary. We know from the past that radiation can arrest, but never cure; this is the opinion of E. A. van Slooten.[34] I would also suggest that if the attempt is made for cure, would it not be easier to remove the nodes than wait until definite signs are available? Again, if we should forget for the time being that being philosophical [2] will not cure cancer, is it not rather absurd to use radiation for treatment in the presence of existing cervical nodes that are palpable but not fixed? In the words of one American surgeon,[32] there are no cases that can not be treated by surgical intervention provided there are enough medical indications and sound surgical judgment to do so. Neck dissection will be done only if palpable nodes are found at the time of operation. It is felt that, if metastasis occurs at a later time, they can still be controlled by surgery or radiation.

We have seen that surgical intervention in itself, when employed justifiably, is the treatment of choice, provided, of course, that all medical questions are well answered. Radiation can be employed with the same good results in cordal tumors, and the words of Clerf,[33] as an otolaryngologist, will bear this statement out. Radiation can furthermore be employed in all cases of noninfiltrative cordal neoplasms. In tumors whose histological study shows undifferentiated cells, it appears to be the consensus that radiation will give just as good results as surgery. We know, however, the chances of cure are poor when the histological cell structure shows differentiation. When we speak of tumors that are inoperable, usually the indications for surgery are lacking in that the tumor is either differentiated or there is presence of metastasis with fixed cervical nodes. The other case is that the patient refuses surgery. Such criteria can never be questioned.

## Conclusions

Prior to 1951, the follow-up of patients after therapy was better in the United States than in the Netherlands. However, at this time the reverse holds true, owing to smallness of the latter country, proximity of patient to the clinic, and the lack of financial embarrassment. At the Institute of Cancer Registration in Amsterdam [17] the

Netherlands government has instituted this agency, started in 1952, for the express purpose of registering the definitely proved cases of cancer. This center serves as the headquarters for seven subcenters. This agency does not deal with active therapy but with follow-up of the registered patients. So far, of the 200 approved hospitals, 150 of them are cooperating to make this agency successful. The agency further gives information as to the total number of cancer cases that have been diagnosed and the morbidity rate of the disease during a given period of time. It is the purpose of this bureau to serve as a check point to determine the diagnosis, treatment, and prodromal period of each case. It will furthermore provide the answer as to when the onset of treatment of each case began. Up until 1953 laryngeal cancer was treated by the ear, nose, and throat specialists without referring these cases to the registration department. However, even though it is not mandatory to register the number of cases, from the figures obtainable so far it appears evident that cooperation is increasing.

In contrast, because of the vastness of the area of the United States, a patient who had been undergoing treatment could not be followed up closely. In certain instances, even though it be a minor point, a patient is rather loathe to have the initial symptoms examined repeatedly in "follow-up" because of the risk that his poor financial means may entail.

A compilation of the existing nomenclatures as an index to therapy shows they are the same in the United States and the Netherlands when analyzed on a comparative basis. However, in certain instances this is disregarded when statistics are reviewed as compiled under the old methods, both in surgery and in radiation.

While each and every case of laryngeal carcinoma should be evaluated in its entity, I believe it is right to categorize each one separately, especially so when diagnosis is concerned and basic therapy employed.

It is my opinion that employing the idea of cordal and extracordal laryngeal tumors as a basis for indicated therapy will improve the understanding of the over-all picture of cure.

When contrasting the average statistics, in so far as the results are obtained either from surgery or from some form of radiation, the statistics presented here can be employed as a check in the final analysis for therapy.

The over-all picture of therapy based on statistical findings in the Netherlands does not compare favorably with that in the United States in a comparable era. However, it is worth while noting that the statistical average of surgical treatment in the extracordal groupings does compare on an equal basis. The discrepancy lies in the cordal group, where laryngofissure was employed. It is hard to evaluate a comparative study of radiation results, as not enough statistics are available in the Netherlands. However, the results obtained from radiation in treatment of true cordal tumors alone in the United States speak for themselves.

The contents of this dissertation were compiled in part through personal interviews and in part from a review of the findings of several authors, the titles of whose articles can be found in the table of references. I wish to apologize to any of the authorities who might be offended by my attempt to fit their data into a standard nomenclature which I have set up to make comparisons more readily understood.

The various chiefs of the otolaryngological services in the Netherlands cooperated in making this paper possible: Prof. L. B. W. Jongkees, director of the service at the University of Amsterdam; Prof. H. A. E. van Dishoeck, director of the service at the University of Leiden; Prof. A. A. J. van Egmond, director of the service at the University of Utrecht, and Prof. E. Huizinga, director of the service at the University of Groningen. Dr. K. Breur, director of the Radio-Therapeutisch Instituut at Rotterdam, cooperated in furnishing advice and information pertaining to the field of radiation in the Netherlands.

W. H. Struben (Arts), chef de Clinique of the Wilhelmina Gasthuis and director of the Ear, Nose, and Throat service of the Antoni van Leeuwenhoek Cancer Institute in Amsterdam also cooperated in making this study possible.

The facts presented in this paper were checked for accuracy by Prof. Jongkees and Mr. Struben.

Translations into English of Dutch articles pertaining to the subject matter were done by Miss P. M. Moore, of the educational department of Scotland, and by L. van Gelder (Arts), of the Wilhelmina Gasthuis in Amsterdam.

My wife, Rosalie Sandoz Henken, B. J., through her journalistic experience, helped make this article more readable.

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Netherlands Cancer Institute.

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# Magnetic Removal of Foreign Bodies from the Esophagus, Stomach, and Duodenum

MURDOCK EQUEN, M.D.; GEORGE ROACH, M.D.; ROBERT BROWN, M.D., and TRUETT BENNETT, M.D., Atlanta

In the past 50 years many foreign bodies have been removed with forceps from the esophagus, and many an open safety pin in the esophagus has been led with forceps into the inflated stomach, there reversed to be drawn back up the esophagus with its point harmlessly trailing. By means of forceps under fluoroscopic guidance a number of things have been also recovered from the stomach.

The introduction of the Alnico magnet and its modifications by Equen made it possible to remove ferrous bodies from the duodenum through the stomach and esophagus. The first foreign body to be pulled up from the duodenum was reported by us [1] in 1947.

Magnetic foreign bodies proximal to the ligament of Treitz, in our opinion, can be most safely and rapidly removed from the gastrointestinal tract with the magnet. We therefore like to remove most of them, if only to spare the parents anxiety.

It is important to select the type of Alnico magnet best adapted to the individual case. The Equen magnet is presented as a cylinder from 3 to 6 mm. in diameter, from 30 to 60 mm. in length; some models are slightly bent near the exploring end. The first one was attached to a wire encased in a Levin tube; at the far end there was a hand bellows for the purpose of inflating the stomach through a hole in the tube near the magnet end. The magnet may be mounted on a flexible stylet, such as a ureteral catheter, for the removal of metallic objects in the tracheobronchial tree. A heavier magnet on a stiffer mount is more valuable in leading an open safety pin down the esophagus into the inflated stomach before hauling it back up.

For all work in the duodenum and for some in the stomach, the bent magnet is attached to a braided-silk, radiopaque string; this string passes through a cap on the straight end of the magnet to make the pull more direct. Such a magnet may be washed down with an ice-cold soft drink which will also help carry it through the stomach, slightly distended by the release of the carbon dioxide.

Before inserting the magnet, adequate roentgenologic studies are required; these include the head, neck, and trunk. Because foreign bodies change their positions and because you need to know what is going on, the operation must be done on a fluoroscopic table. Though in a recent 12-month period, 158 children were brought to the Infirmary with a story of having swallowed some metal object which x-ray failed to reveal, attention should always be paid to such stories. Even in the absence of such a story, in the presence of persisting difficulty in swallowing, the child is entitled to an x-ray investigation.

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From the Ponce de Leon Ear, Nose and Throat Infirmary.

Presented by Dr. Equen before the Sixth International Congress of Otolaryngology, Washington, D. C., on May 9, 1957.

Most of the material in this paper is from the Dr. Equen's monograph, Magnetic Removal of Foreign Bodies from the Tracheobronchial Tree, Esophagus, Stomach and Duodenum, Charles C Thomas, Publisher, Springfield, Ill., April, 1957. Figures 1A, 1B, 2A, 2B, 3, 4, 5A, 5B, 7, 9A, 9B, 10, and 11 appeared in this monograph. Figure 7 also appeared earlier, in Equen, M.: Magnetic Removal of Foreign Bodies from Food and Air Passages, Arch. Otolaryng. 46:225, 1947. Figures 6A, 6B, and 8 have not hitherto been published.

A Berman locator will often serve to determine if the foreign body is or is not magnetic.

The perfect relaxation afforded by a general anesthetic seems to us safer for the patient, though occasionally in well-poised adults we do without it. In our clinic, vinyl ether is usually used for the induction stage, and then a switch is made to sulfuric ether.

A tracheotomy tray, laryngoscopes of various sizes, and other endoscopic instruments should be available in case of need.

Perhaps the most important thing for the smooth functioning of magnetic operations, as of so many other delicate procedures, is to have an experienced team accustomed to working together.

The further discussion of this subject will be simplified by taking up the organs individually.

### The Esophagus

When a metallic foreign body is arrested in the esophagus there is no question that it should be removed. The most dangerous foreign body encountered here is the open safety pin. When such a pin gets well beyond the cricopharyngeal constriction, the spring is of necessity down, the point up: attempts to pull it back up the esophagus may thus cause perforation of the wall of the esophagus and perhaps severe laceration; similar damage may be done if the patient tries to vomit up the pin.

When the pin is relatively small, however, it is often possible with forceps to pull the point of the pin into the esophagoscope and so retrieve it; the keeper end of the pin will do no damage to the elastic esophagus. It is possible either to cut the pin in the esophagus or to close it; once the pin is cut or closed, its removal is easily accomplished, but the first steps in each of these methods is technically difficult and full of danger to the patient.

Another standard method is to grasp the ring with forceps, lead it into the inflated stomach, and there, under fluoroscopic guidance, reverse the pin so that, when it is drawn back up the esophagus, the point will trial behind innocuously. The chief objection to this method is the uncertainty of reversing the pin in the stomach; moreover, in attempt-

Fig. 1A.—The magnet has led the open safety pin into the fully inflated stomach of a girl of 9 months.

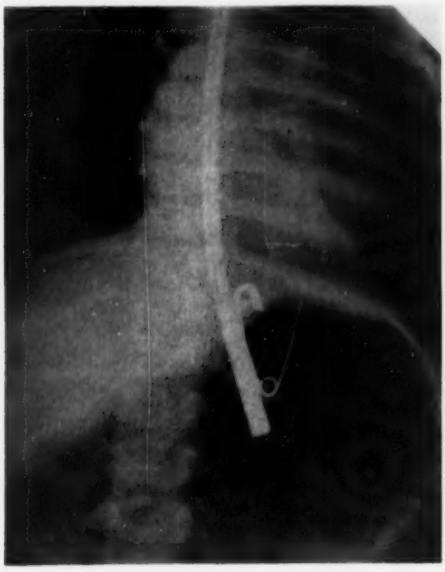

Fig. 1B.—The magnet is now pulling the open safety pin up the esophagus without risk of puncture or laceration.

ing to do so, there is a possibility that the pin may be dislodged from the forceps.

When a heavy old-fashioned steel diaper pin is open in the esophagus, we are confident that the simplest and safest way to remove it is with a relatively large, straight magnet mounted on a heavy, semiflexible stylet. With this one leads the pin into the inflated stomach, where, under fluoroscopic guidance always, one jiggles the oral end of the stylet until he sees that the pin is reversed. Then he draws the magnet and pin back up the esophagus, the lethal point lagging safely behind (Figs. 1A and 1B).

In all, 41 cases of open safety pins in the esophagus have been treated by us without the magnet. In two fatal cases the pin had perforated the esophagus before the child arrived in Atlanta. In a third, attempts to reverse the pin in the stomach were unsuccessful. In others the pin was recovered by one or more of the methods outlined above.

In 14 cases, a large open safety pin has been removed with a magnet. Eleven of these patients were under 3 years, but one was 73. Most of them went home within 36 hours.

The carpenter's heavy staple is as dangerous as the open safety pin: like the pin it goes down with its harmless rounded end first, its points up. We like to remove them from the esophagus (Figs. 2A and 2B) as we do safety pins. Fortunately, however, the staple, unlike the safety pin, is not often within the reach of children in the diaper age.

We have removed 14 other metallic objects from the esophagus with the magnet.

### The Stomach

Of course the majority of foreign bodies reaching the stomach will pass through the gastrointestinal tract uneventfully. It has been most surprising to us to see how many straight pins, even open safety pins and staples, have done so in the Infirmary a day or two after their ingestion! We cannot therefore quarrel with those who advocate waiting a day or two to allow the forces of nature to propel the foreign body onward, provided it has no sharp point or cutting edge. In such cases we may even allow the parents to take the child home after impress-

Fig. 2A.—A staple, like an open safety pin, must travel down the esophagus with its points up. The patient was a Negro infant.

Fig. 2B.—A curved magnet on a fairly stiff stylet had led this staple into the greatly distended stomach, there reversed it, and it is now pulling it back up the esophagus.

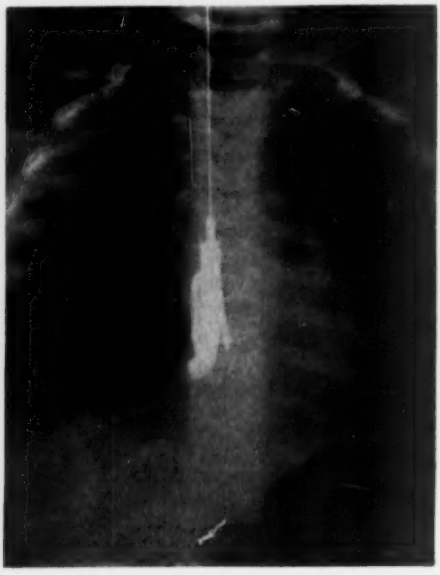

Fig. 3.—Lateral view of magnet in contact with four bobby pins in the stomach of a little girl of 2 years. Contrast this with the laterals of foreign bodies in the duodenum.

Fig. 4.—Lateral view of magnet in contact with a 5 cm. nail in the stomach of a boy of 4 years. Note how far it is from the spine.

ing upon them the importance of watching the stool for the reappearance of the foreign body, and explaining to them the signs of perforation and of obstruction. The difficulty is in getting the parents to accept this conservative proposition.

Fig. 5A.—The bent magnet is surrounded by four straight pins in the stomach of a boy of 4 years.

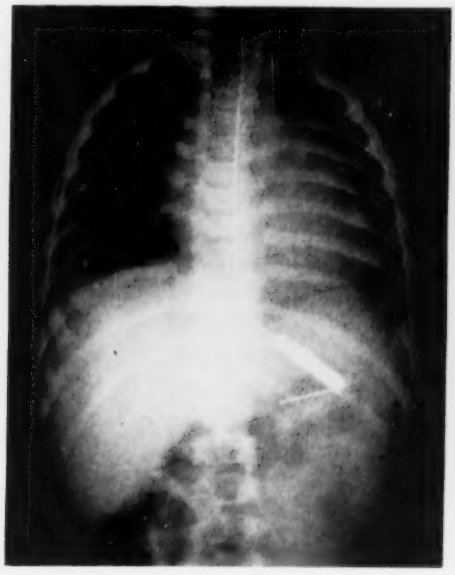

Fig. 5B.—The magnet is about to deliver the four pins into the esophagoscope. We were surprised that the cardiac sphincter did not strip a single pin from the magnet, but to expect to get them all by the cricopharyngeus seemed to be putting an undue strain on our luck.

Fig. 6A.—Lateral view of a corsage pin in the stomach of a child of 3 years. It looks deadly.

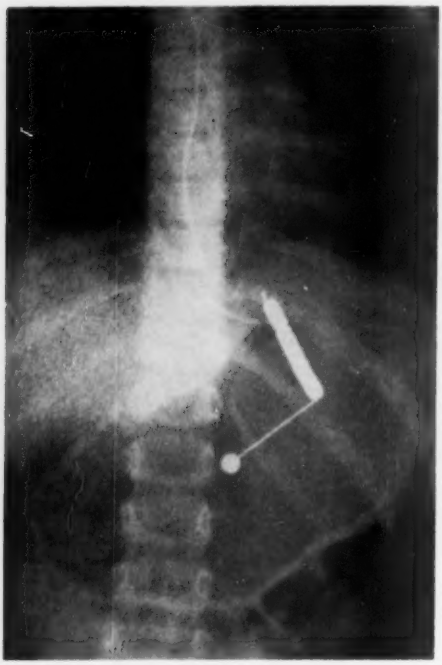

Fig. 6B.—Posteroanterior view of the corsage pin in the greatly distended stomach. Its point adheres to the end of the magnet.

On the other hand, one can never tell when even a straight pin will result in disaster. We have never lost a case in which magnetic removal of a foreign body from the esophagus, stomach, or duodenum has been attempted, so we tend to go after most of them.

At the Ponce de Leon Infirmary in 189 cases foreign bodies have been removed from the stomach with the Equen magnet. Of these 189 patients, 102 were not more than 3 years old; the exact age of 14 was not recorded, but most of them were in the same age group. There were 51 patients ranging from 3 to 7 years, 15 from 8 to 15, and 7 were adults. They were rather evenly divided as to sex.

The bobby pin was the chief offender, being found in 45 cases; in 3 of these there were 2, 3, and 4 (Fig. 3) pins respectively.

There were 31 cases of nails (Fig. 4), 15 of closed safety pins, and 14 of straight pins (4 pins in one stomach: Figs. 5A and 5B), 5 open safety pins, 5 heavy staples, 2 corsage pins (Figs. 6A and 6B), and a wide assortment of other things, mostly small toys or parts of them.

When one wishes to remove a foreign body that responds to the Berman locator from a child's stomach with a magnet, he has his choice of two techniques: he may use the original stomach magnet, or, better, one with a bend. Once such a mounted magnet is in the inflated stomach, under the guidance of a fluoroscope, it is but the work of seconds to approximate the magnet to the foreign body and then to remove it. Or one may be content to have the child swallow a bent magnet attached to a radiopaque string, promoting distention with a carbonated drink, and check the progress of the magnet with the fluoroscope. The child himself may announce when contact has been established between the magnet and the pin.

The stomach has extraordinary tolerance for steel objects. We have removed one large lock cover that had remained six weeks

in the stomach of a child of 5 years without causing any symptoms—except to her parents. This case was of unusual interest in yet another way: though a standard magnet attracted it, it was not strong enough to bring it up, so a larger magnet was obtained, and with this success was achieved.

Data on 189 cases of foreign bodies removed from the stomach with the Equen magnet have been presented. This series may be the largest on record of foreign bodies drawn out of the stomach from above in one institution. It is emphasized again that the record is unmarred by a single fatality. Occasionally the patient was in the hospital only a few hours, usually one or two days, never more than three days, after operation.

## The Duodenum

The x-ray is indispensable in locating a radiopaque foreign body in the gastrointestinal canal, though often more than one plate is necessary. If the bobby pin, for example, lies to the right of the vertebral column, roughly parallel, but with its upper end slightly further from the spine, it is probably in the descending duodenum. If the position is somewhat transverse, one end to the right and the other pointing upward and across the spine, it should be in the third loop.

If the plate shows the foreign body transversely across the spine, it suggests that it is within the duodenum. If, however, the patient was erect when the plate was made, and the stomach is ptosed, the foreign body may lie horizontal, across the lower lumbar vertebrae (Fig. 7). In a small child the stomach is physiologically high and transverse in the abdominal cavity and even when he is lying on his back, the foreign body must enter the pyloric antrum. When you

Fig. 7.—Most of our plates are made with the patient in the recumbent position. In this position gravity will pull the metallic foreign bodies to the fundus. At the first blush these three bobby pins appear to be in the transverse portion of the duodenum. They lie below the third lumbar vertebra. This adult was in an erect position at the time the plate was made, and further studies proved the pins were in the stomach, from which they were recovered with a magnet.

Fig. 8.—Though this boy of 18 months was in the recumbent position, further studies showed that the bobby pin was still in his stomach. The open end of the pin appears to be (and is) surrounded by intragastric air. The pin lies just below the body of the 12th dorsal vertebra.

Fig. 9A.—With the child recumbent, the fundus of the stomach is the lowest part and the magnet has been drawn by gravity to this part. The bobby pin remains in its original position across the vertebral column, where it had been held up three days before admission to the Ponce de Leon Infirmary.

Fig. 9B.—The magnet in the descending limb of the duodenum has caught up with the bobby pin and is about to remove it. The radiopaque string beautifully demonstrates the first portion of the duodenum.

see a nail or bobby pin across the upper lumbar vertebrae of a youngster then, it may still be in the stomach (Fig. 8). Sometimes, though, when the child has just lain down, the foreign body may remain briefly across the spine, especially if there is little air in the stomach; rocking him back and forth will help clear up uncertainty.

The law of gravity is immutable: if the stomach is moderately distended, the foreign body will fall to its lowest part. If the patient is lying on his back, the lowest part will be the fundus. Therefore, if you see the magnet in the fundus and the foreign body across the spine some distance away from the magnet, you can be sure that the foreign body is beyond the pylorus (Fig. 9A), but without further studies you cannot be sure that it is still in the duodenum.

Foreign bodies in the duodenum may be suspected from simple posteroanterior plates. Such locations can be rendered fairly sure with a lateral view: Figures 10 and 11 show a sign first noted by us. The opaque string from the magnet to which the foreign body is attached, the slack having been taken up, goes forward (in the lateral view) through the pylorus and pyloric antrum; once having cleared the protrusion of the posterior wall of the stomach caused by the pancreas and vertebral column, it appears to turn sharply backward and slightly up, hugging the lesser curvature before passing through the cardia into the esophagus. In the posteroanterior view the string shows a wide sweep (as in Fig. 9B), as it would in a transverse roentgenogram at this level if such were possible. Such a plate as Figure 10 or Figure 11 proves that the foreign body is in the duodenum.

The fluoroscope is invaluable in duodenal work. We have seen foreign bodies arrive in the duodenum within an hour of being swallowed; we have seen them in the duodenum 17 days after being swallowed. We have seen the magnet catch up with the foreign body in 2 hours, and fail to make contact for more than 20 hours. We have seen a foreign body pass into the duodenum

Fig. 10.—The magnet and nail are in the descending limb of the duodenal loop of an infant of 4 months. Air in the colon is immediately anterior to them. The radiopaque string passes almost on a level (anteriorly in this diagonal view) through the first portion of the duodenum and pylorus, into the gas-distended stomach. It then goes backward and upward through the cardia into the esophagus. The string had been somewhat tautened at the time of the plate.

Fig. 11.—The magnet is seen head-on in the transverse part of the duodenum. (Note again the course of the radiopaque string.) It has made contact with the nail, which is seen in the ascending loop. The little girl was 16 months old.

on the table and, in the induction stage of anesthesia, we have seen one regurgitated from the duodenum back into the stomach.

It is difficult to estimate which metallic foreign bodies will get through the duodenum and which will not. It is in large measure a matter of relativity. A 6 cm. nail or a bobby pin may pass through the duodenum of an adult male quickly and uneventfully, but such an object can hardly be expected to get through the convolutions of the duodenum of a baby a few months old.

If a foreign body becomes impacted in the duodenum it can only be removed by use of the magnet or through an open operation. Such an operation requires section of the abdominal wall, and then the surgeon must grasp with his hand the relatively small, though thick-walled, retroperitoneal duodenum. Then he must cut through the wall of the gut, remove the foreign body, and sew up the incision. Because of the disproportion between the size of the adult hand and the size of the upper abdomen of the small child, it is a difficult procedure and one that carries considerable risk. At best the child must be given intravenous fluids for several days, no mean undertaking in the case of infants, and he must remain in the hospital a week or more.

In contrast, the magnet may be expected to retrieve the foreign body in a very brief space of time, and the child can usually go home in a couple of days.

In 19 cases at the Ponce de Leon Infirmary metallic foreign bodies have been recovered from the duodenum with the magnet. Thirteen patients were 3 years old or younger; none was older than 6. There were 7 boys and 12 girls. Of the foreign bodies there were nine bobby pins, eight nails, one hairclasp and one straight pin. Each of these cases has recently been reported in some detail.

It may be remarked again that in one case the ordinary intraluminal magnet was not strong enough to budge the nail; a 7½ lb.

Alnico magnet was applied on each side of the thorax until the nail was dislodged.[2]

In two cases the foreign body obstructed or perforated the duodenum, and it was necessary to resort to open surgery. Both children, we are thankful to say, recovered.

There are two principal reasons for failure of an Alnico magnet to recover metallic objects from the gastrointestinal tract: the first and most common is that the foreign body is not magnetic, and the second is that the magnet does not succeed in overtaking the foreign body. Again individual decision is necessary. If the foreign body clears the ligament of Treitz, we have found it is impossible to retrieve it with a magnet. Once it has entered the jejunum, the next danger point is in the region of the cecum: we know of several cases where a straight pin, for example, has perforated the wall of the intestine to be the focus of an abscess.


## Summary

With the Equen magnet we have recovered 14 open safety pins and 14 other objects from the esophagus; in 189 cases we have removed foreign bodies from the stomach, and 19 foreign bodies from the duodenum. There has been no death in the series. In two other cases perforation of the duodenum required abdominal section, but both children recovered.


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# Excision and Pari-Passu Repair of Pharyngeal Diverticula

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In the West Riding of Yorkshire, as elsewhere, the incidence in the community of pouches opening between the ascending and horizontal fibers of the inferior constrictor of the pharynx is about 1 in 100,000.

Such formations are probably atavistic in origin. Van den Wildenberg [1] recalls that similar peculiarities have been demonstrated in certain mammals, such as elephants and camels: many problems concerning man's vertebrate inheritance remain to be elucidated as science unfolds.

In 1767, Ludlow [2,3] first described a bag formed in the pharynx. For long afterwards there was every justification for medical men to advise against operative intervention, despite the miseries of regurgitation and the extremities of starvation to which the patients were condemned. The first successful excision was performed by Wheeler [4,5] in 1886. Mortality remained high. The two-stage operation of Goldmann [6] in 1907 and the diverticulopexy of Hill [7] in 1926 are foothills, bearing beacons in their day, which most contemporary surgeons have had to surmount in their pilgrimage. Archaic as present-day methods may one day seem, excision is now one of the most satisfactory procedures in the whole gamut of surgery. Its elegance is enhanced if excision and repair are carried out step by step, alternately and equally.

My five male and one female patient had experienced dysphagia and regurgitation for many years. Most were hypertensive. All made good recoveries.

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Otolaryngologist to the Hospitals of Halifax and Huddersfield.

Recently my colleague Miss K. M. Watson has been combining nitrous oxide anesthesia with Fluothane (2-bromo-2-chloro-1, 1,1-trifluorethane). The last patient could have walked back from the operating room if he had been allowed to do so.

With a competent assistant (such as Dr. Ishrat Husain) to retract the sternomastoid and present the parts for surgical treatment, and with an experienced sister to hand the sutures and instruments, it was found that the operating time could be cut down from about two hours to about one hour. In most cases, the neck was infiltrated with 1:20,000 epinephrine solution, unless this was felt to be contraindicated. An incision was made from a point a fingerbreadth above the right sternoclavicular joint to one 3 fingerbreadths above the center of the left clavicle. The left anterior jugular vein was ligated in continuity and divided. The left external jugular vein was guarded during exposure of the anterior border of the sternomastoid by blunt dissection, to obviate any risk of air embolism. The anterior belly of the omohyoid was divided between ligatures to include any small vessels which might produce a postoperative hematoma. The position of the thoracic duct was borne in mind and the line of the recurrent laryngeal nerve identified sufficiently to avoid approaching it too closely. The inferior thyroid artery was sought out and divided between ligatures.

The diverticulum, now in view, was elevated from the mediastinum by blunt dissection, separation being made in the optimal layer of loose areolar tissue. It was never necessary to split the sternum. The cavity thus evacuated, anterior to the prevertebral

Fig. 1.—A pharyngeal diverticulum.

fascia, was then lightly packed with ribbon gauze, to avoid contamination from above, during the subsequent stages.

A traction suture was next inserted above the neck of the pouch, which was excised, and the pharynx was repaired in a series of equal steps, interrupted only by the insertion of a little finger into the esophagus, after which the glove for that hand was changed. No perceptible stricture was found below the neck of the diverticulum in any of the six cases. During this almost simultaneous repair the sutures were passed from the mucosa to the muscularis of one side of the ellipse and from the muscularis back to the mucosa of the other, so that the knots lay in the lumen. A second row of sutures, passed through the muscular coat only, invaginated the first row. Fine silk or absorbable surgical suture was employed; as a Leeds graduate, I incline toward absorbable suture materials.

After removal of all gauze packs, penicillin and streptomycin powder was dusted into the cavity and a strip of corrugated rubber inserted through a stab wound in the skin, on through the gap between the sternal and clavicular heads of the sternomastoid and down into the mediastinum; it was left in position for 48 hours. The wound was then closed.

Throughout the operation and more especially at its conclusion, care was taken to keep the contents of the diverticulum out of the air passages. One pouch, when opened, was found to contain the sinister warning for future cases of five orange pips. No feeding tube was inserted into the

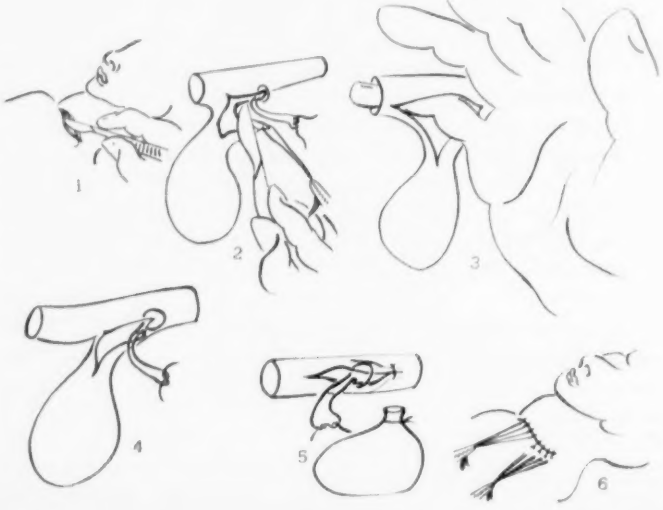


Fig. 2.—Simultaneous excision of a diverticulum and repair of the pharynx. The pharynx is sutured so that the first line of knots lies in the lumen and the second rests on the prevertebral fascia.

pharynx and esophagus, as a rule. Patients found it easier to swallow semisolid food at first.

The technique described above is but one aspect of a system of primary restoration of particular value in the treatment of injuries and tumors in the vicinity of the maxillary antrum. Rhinoplasty and tympanoplasty afford many further illustrations of the manner in which more perfect healing may be obtained than by secondary plastic procedures. Again, in laryngectomy, as the cartilaginous skeleton of the larynx, containing the tumor, is being removed, progressive restoration of the pharynx is conducive to meticulous suturing of the delicate mucous membrane of the pharynx, leaving all the knots of the first layer within the lumen. Thus fistulae are avoided. To build up what has been broken down as one goes along yields the best results.

## Summary

In six advanced cases of pharyngeal diverticulum a methodical plan of operation was developed. Excision and repair were carried out alternately, in a series of equal steps. This was found conducive to an elegant technique and to prompt healing. The earlier the reference, the less the risk.

Green Leas, Savile Park.

## Addendum

At a meeting of the Leeds and West Riding Medico-Chirurgical Society on Oct. 4, 1957, the above contribution was briefly presented. Transparencies in color of the operation were projected. That pharyngeal diverticula were inherently septic or usually harbored saprophytic micro-organisms was queried but it was agreed that preliminary lavage with 2% sodium bicarbonate was advisable.

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Therapy in Otolaryngology

# Use of Corticosteroids in Otolaryngology

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"Corticotropin and the related adrenocortico-steroids are the most powerful agents that have come into man's possession since Prometheus stole fire from the gods. But their power can be for evil as well as for good, and we are only beginning to know their possibilities and adapt them to our use." This statement was made before the American Laryngological Association a few years ago by Dr. Richard Kern,[1] and its significance should be recognized by all otolaryngologists.

To properly appraise the steroids it might be well to point out briefly the physiologic effects of corticotropin (ACTH). The adrenal cortex in response to stimulation by corticotropin from the anterior pituitary produces at least 28 steroid substances. The outer layer, or zona glomerulosa, produces desoxycorticosterone (DOC) and is concerned with salt and water balance. The inner layer, or zona reticulosa, has to do with gonad function and is the source of estrogen, androgen, and progesterone. The middle layer, or zona fasciculata, produces hydrocortisone and probably aldosterone and has an influence on the metabolism of carbohydrates and proteins.

It is the stimulation of the steroids from the middle layer, principally hydrocortisone, which is responsible for the effect of corticotropin on the allergic manifestations of hypersensitivity. Local swelling and itching are diminished, and there is a marked modification of the inflammatory reaction. The clinical usefulness of corticotropin and cortisone is due to their effects in altering tissue reaction to hypersensitivity and inflammation, and through this altered reaction such phenomena as allergic rhinitis, allergic sinusitis, nasal polyposis, and asthma can frequently be controlled.

The exact mechanism of action of these hormones is not definitely known, but Rawlins[2] has pointed out that there are three actions of these corticosteroids that are of importance to the otolaryngologist. These are (1) anti-inflammatory action; (2) action in regard to infection; (3) action on capillary and connective tissue permeability.

## Anti-Inflammatory Action

It has been demonstrated that inflammation as such always occurs in the connective tissue, and when one examines an inflamed nose, whether of allergic, infectious, or traumatic etiology, the swelling is not in the epithelium but in the edematous ground substance of the connective tissue under the epithelium. It is a type of chain reaction in the connective tissue which is the result of stress. Stress produces cell damage which sets off and continues the inflammatory reaction. The damaged cells are principally fibroblasts and mast cells, and the released substances are histamine, hyaluronidase, heparin-like substances, etc. Local inflammation and generalized stress differ only in degree and localization of the stress.

The corticosteroids depress all of these processes.

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### Action in Regard to Infection

The glucosteroids in large doses impair the host's resistance to many infections, while in very small doses or with short-term therapy there may be an improvement in the host's resistance to infection.

### Action on Capillary and Connective Tissue Permeability

The pericapillary sheath is a structure of great importance in relation to edema. It is a condensation of fibrils and very firm ground substance which supports the capillaries. When it is firm, it prevents edema. When it is liquefied by hyaluronidase, edema occurs.

The corticosteroids help to keep this structure firm and prevent edema.

These hormones thus effect a rapid improvement in the symptoms of many diseases without influencing the cause, which must be eliminated if one wishes to obtain a lasting result.

In none of the many disorders in which steroid therapy has had any effect has there been shown to be a deficiency of the normal output of corticotropin or hydrocortisone, and consequently such treatment is not replacement therapy but is based upon doses much greater than the amounts of these steroids normally present in the body and is therefore potentially dangerous.[3]

Of basic importance in the use of steroid therapy is a knowledge of the effect of the administration of an endocrine hormone upon the normal glands of the recipient. Even the least amount has some effect—the degree depends upon the amount and the duration.

When corticotropin is given to a person, his own anterior lobe of the pituitary promptly produces less than it did. If the dose of corticotropin exceeds 1 unit a day, the man's own gland stops making corticotropin entirely. For a week or 10 days this does no obvious harm, but after that atrophy begins.

If a person receives corticotropin in doses of more than 1 unit per day for three months, then his own anterior lobe will have undergone obvious atrophy. During this same time the excessive stimulus will cause the adrenals to hypertrophy, and so after three months they are three or four times normal size. It takes at least six months after cessation of therapy for the anterior pituitary to resume its normal function and size.

If hydrocortisone is given in a 12 mg. daily dose for 10 days, the anterior pituitary does not put out any corticotropin and after three months there is an atrophy of marked degree. At the same time the adrenal cortex stops functioning and after three months has shrunken to one-third or less its original size.

A common fallacy is the belief that prednisone and prednisolone do not have this effect since only 5 mg. daily may be needed to keep the patient comfortable. A 5 mg. dose of one of these substances is comparable not only in symptomatic relief but in production of glandular atrophy to 15-20 mg. of hydrocortisone. Armour Laboratories sent a warning to physicians last year stating that "while the new steroids are a valuable therapeutic addition our concern deals with a false sense of security which appears to prevail among physicians who had previously shied away from corticoid therapy. A great many physicians evaluate safety in steroid therapy essentially in terms of side actions such as sodium retention, weight gain, hypertensive effects, decrease in sugar tolerance, etc. These side effects are really early warning signals of beginning overdosage."

One effect, however, adrenal cortical suppression and atrophy, is not an overdosage effect, but the unavoidable accompaniment of any corticoid therapy whether cortisone, hydrocortisone, prednisone or prednisolone is employed.

The indications for treatment with the corticosteroids resolve themselves into short-

term therapy (10 days or less), long-term therapy, and topical application.

In otolaryngology the conditions for which short-term therapy is indicated and useful are as follows:

1. Status asthmaticus
2. Acute edema of the larynx
3. Penicillin and other severe drug reactions
4. Severe hay fever
5. Midline granuloma of the facial tissues
6. Erythema multiforme
7. Pemphigus
8. Contact ulcer of the larynx
9. Laryngeal polyps
10. Extensive nasal polyps
11. Fresh stricture of the esophagus
12. Bell's palsy
13. Local spray of hydrocortisone in respiratory allergy

The dosage of these hormones is still unstandardized. An attempt should be made in each case to determine the least amount capable of maintaining clinical improvement. A suitable dosage of cortisone initially for the average adult patient is 100 mg. daily in divided doses. It may be given orally, although the intramuscular or intravenous route can be used. Two types of corticotropin are available. One is the aqueous suspension every six hours and the other is a suspension in gelatin-containing medium. With this latter preparation only one or two injections daily may be required. An average daily dosage of either drug is 100 mg.

The use of these hormones in large doses over long periods of time is not to be recommended in the practice of otolaryngology. The long-term effects of long-term use of corticosteroid therapy are as follows:

1. Atrophy of the anterior pituitary and adrenal glands
2. Hypercorticism
3. Hypertension
4. Edema due to salt and water retention
5. Mental symptoms, particularly in those with previous psychoses
6. Convulsions
7. Glycosuria
8. Peptic ulcer
9. The possibility that cleft palate may arise in the fetus while the mother is on steroid therapy
10. Addiction

Topical application of these hormones is without dangerous side-effects inasmuch as the concentrations absorbed from intranasal hydrocortisone spray in allergic rhinitis or polyposis or hydrocortisone ointments in eczema of the ear are practically nil.

In a one-year study which I[4] conducted a few years ago on the intranasal use of hydrocortisone alcohol in 174 patients with allergic rhinitis and allergic rhinitis with nasal polyps I concluded that not only does a topically applied hydrocortisone alcohol solution exert a beneficial effect on the allergic nasal mucous membrane but also that it can exert this effect even when given in extremely dilute solutions. The beneficial effect of the steroid on polypoid tissue was frequently marked and occasionally dramatic; however, it was not always permanent. It was observed that when multiple polyps were present the posterior ones shrank less than the anterior ones, indicating a local action of the steroid. Cortisone as well as various concentrations of hydrocortisone were used by the patients in this study, and the greatest objective as well as subjective improvement was noted with a combination of 20 mg. per 100 cc. of hydrocortisone alcohol solution plus two vasoconstrictors.

Myers recently reported at the International Congress of Otolaryngology in Washington on his experiences in the treatment of allergic nasal polyps by intrapolyp injection of long-acting hydrocortisone (tributyl alcohol) and prednisolone (tri-butyl alcohol). He stated that he had good results without side-effects and pointed out that the nasal mucous membrane is capable of regression to normal or near normal after having undergone polypoid change. Other authors have reported serious side-effects from the direct injection of the corticosteroids into nasal polyps.

The contraindications to oral, intravenous, or intramuscular steroid therapy in otolaryngology are as follows:

1. Acute infections for which there is no available effective antibiotic

2. Chronic infections, such as tuberculosis
3. Peptic ulcer
4. Chronic liver disease
5. Severe hypertension
6. Diabetes
7. Previous psychosis
8. Pregnancy
9. History of breast or genital carcinoma in females.

## Conclusions

1. Corticosteroids have a definite place in the treatment of otolaryngologic conditions which require short-term therapy or topical treatment.

2. Intranasal hydrocortisone alcohol spray is efficacious in the treatment of seasonal or perennial allergic rhinitis and allergic rhinitis with polyps.

3. Treatment of hay fever with the corticosteroids other than topically is like shooting sparrows with atomic warheads, as Kern recently stated.

4. Corticosteroid therapy alone is never curative.

5. Corticosteroid therapy is always potentially dangerous.

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# Effective Antibiotic Therapy

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In the Wherry Memorial Lecture, entitled "Res Medica 1956," published in the March-April, 1957, issue of the *Transactions of the American Academy of Ophthalmology and Otolaryngology*, Arthur Proetz[1] made the following statement: "Unquestionably the greatest single advance in medical therapy in the 15 years since Wherry lived—indeed in the 60 years since Pasteur lived—is the antibiotic in its various forms." In contrast to this statement, it does seem odd that in the list of more than 250 papers presented at the Sixth International Congress of Otolaryngology in Washington in May, 1957, there was only one paper dealing with antibiotic therapy. After consideration of these two facts, one comes up with two ideas. First, either that the principles of antibiotic therapy are clear-cut, well-defined, and hence not worth talking about, or, second, that otolaryngologists simply aren't interested in discussing or knowing what is the best type of antibiotic therapy for the various types of infection we are called upon to treat. Because I think both inferences are incorrect, I propose to take the next 15 minutes to discuss antibiotic therapy.

The principles of effective antibiotic therapy were well described in 1946 by Sir Alexander Fleming[2] in his book entitled "Penicillin: Its Practical Application." This book and its second edition,[3] published in 1950, are still important references and will help anyone to evaluate the conflicting claims made in the flood of multicolored antibiotic advertisements overcrowding the mailbox of every physician.

Sir Alexander's guiding principles apply to the newer antibiotics as truly as they do to the use of penicillin. One can best evaluate the claims made by the vendors of the more recent antibiotics if he recalls Sir Alexander's "General Rules." One has only to substitute the name of the antibiotic in question for penicillin. The rules are as follows:

1. Penicillin should be used only when there is an infection by a penicillin-sensitive microbe.
2. Penicillin must be administered in such a way that it comes in contact with the infecting microbe.
3. The dose should be such that in the *infected area*, the concentration of penicillin is sufficient to *destroy* the bacteria.
4. Treatment should be persisted in until the infection is defeated.

I should like to make a few comments regarding these rules. Rule 1. We know at the present time there are many strains of Gram-positive cocci which have become resistant to the action of penicillin. Thus it seems obvious that sensitivity cultures should be used for all serious or potentially serious infections. In 1945 our laboratory first began using sensitivity cultures made on blood agar plates, incorporating three concentrations of penicillin. Recently we have been using the disc sensitivity culture method advocated by McMahon,[4] because the laboratory report can be obtained within 24 hours.

Rule 2 makes it evident that systemic administration of the antibiotic is infinitely superior to topical application. In all but the most superficial infections the organisms are in the tissues where they can be reached only by diffusion of the drug carried by the blood stream. Fortunately the unsatisfactory results of the topical application, including

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aerosol administration, have been recognized, so this method has been discarded by most men.

Rule 3 stresses the fact that for all infections other than bacteremia, it is not the blood level that is important, but the quantity of drug that has diffused into the infected area. I agree with Eagle and associates,[5] who say, "A large dose may promote penetration of penicillin into walled-off foci of infection which are only slowly equilibrated with the tissue fluids, and into which penicillin might not diffuse in adequate concentration after a small dose which provides a low head of pressure for a relatively brief period." If one thinks only of the blood levels produced by a recommended dose and of the alleged degree of in vitro sensitivity, he will probably give his patient ⅕ or 1/10 the optimum dose, because he has failed to take into account the probability of poor diffusion into tissues already blocked by edematous fluid. I italicized the word "destroy," because I think too often there is insufficient attention paid to the difference between bactericidal and bacteriostatic amounts of any antibiotic. Admittedly, if the patient's resistance is good, small bacteriostatic doses may be adequate, but if one is dealing with a potentially serious infection, I think it is better judgment to use bactericidal doses. In clinical evaluation of what is a bactericidal dose, it can be said that if one is giving an adequate amount of the right antibiotic the clinical signs and symptoms of the infection will be practically eliminated within 48 hours.

In our experience, Rule 4 is the one most often violated. Many times, so-called relapses occur because use of the antibiotic was discontinued the day the temperature reached normal, perhaps on the second or third day. Dr. Fleming's advice in 1946 to continue use of penicillin for 72 hours after apparent cure of the infection is still sound advice and applies to use of the other antibiotics.

Medical progress is fortunately measurable in figures for morbidity and mortality.

TABLE 1.—*Deaths from Complications of Otolaryngological Infections from 1932 to 1956 Inclusive*

| Presulfonamide Period | | Sulfonamide Period | | Penicillin Period | |
|---|---|---|---|---|---|
| Yr. | Deaths | Yr. | Deaths | Yr. | Deaths |
| 1932 | 6 | 1939 | 3 | 1945 | 1 |
| 1933 | 7 | 1940 | 3 | 1946 | 0 |
| 1934 | 8 | 1941 | 4 | 1947 | 3 |
| 1935 | 4 | 1942 | 0 | 1948 | 1 |
| 1936 | 7 | 1943 | 0 | 1949 | 0 |
| 1937 | 1 | 1944 | 1 | 1950 | 3 |
| 1938 | 8 | | | 1951–54 | 0 |
| | | | | 1955 | 1 |
| | | | | 1956 | 0 |
| Total | 41 in 7 yr. or about 6 per yr. | Total | 11 in 6 yr. or about 2 per yr. | Total | 9 in 12 yr. |

Table 1 shows the mortality statistics for the Department of Otolaryngology of one 300-bed hospital. Our service has approximately 1000 admissions per year. It is notable that there has been only one death due to infection since 1950. It was due to a cerebellar abscess secondary to neglected mastoiditis in a patient who was moribund at the time of admission. Reduction of morbidity and reduction in the number of operations necessary for drainage of pus have paralleled the decrease in deaths due to complications of otolaryngological infections. From the data presented it seems obvious that penicillin by intramuscular injection is superior to the sulfonamides. In our experience the "mycins" have not been as effective as penicillin by intramuscular injection in treating serious infections due to penicillin-sensitive organisms. In vitro tests to determine the sensitivity of the infecting organism can be helpful, but they are not a substitute for clinical judgment. Now more than ever, the otolaryngologist must know bacteriology as well as the advantages and limitations of the available antimicrobic agents.[6]

The conflicting statements found in the commercial literature advertising antibiotics do not simplify the clinician's problem in deciding the relative merits of the available antibiotics. I suggest the 1957 edition of "New and Nonofficial Remedies" evaluated by the Council on Pharmacy and Chemistry of the American Medical Association as a dependable source of information.

For performance of routine sensitivity cultures, our laboratory is at present using antibiotic discs treated with penicillin, streptomycin, chloramphenicol, erythromycin, tetracycline, and novobiocin. McMahon [6] finds disc sensitivity cultures at least 95% as dependable as the methods utilizing pure cultures which require three days to complete. However, to depend on these cultures one must know the actual amount of antibiotic contained in each disc. Greenberg and associates [7] have called attention to the fact that the potencies of most commercial discs are not in accordance with the labeled claims.

The following is a quotation from an editorial [8] in the *New England Journal of Medicine:* "The opening remarks at the Fourth Annual Symposium on Antibiotics held recently in Washington called attention to the 'distinct and dynamic trend toward combined antibiotics' and suggested that a new era in the new antibiotic age may be beginning. . . . The trend toward production, sale and use of antibiotic combinations cannot be denied since it is being so energetically and aggressively pursued by some manufacturers and so often uncritically accepted by many physicians, who readily fall prey to the psychologic warfare embodied in the high-powered techniques of modern sales promotion." The closing line in this editorial is: "Caveat emptor!"

Recent advertising claims much virtue in the use of mixtures of antibiotics. Dowling,[9] after critically analyzing the data, comes to the following conclusions: "The administration of combinations of antibiotics is indicated only in a few highly selected conditions. However, they have been advocated and used in other conditions in which they not only do no good, but actually may be harmful. When combined therapy of antibiotics is indicated, the antibiotics are best given separately, except in the case of mixtures for local application to the skin." A recent editorial [10] in the *A. M. A. Archives of Internal Medicine* also decries the senseless prescribing of antibiotic mixtures. If the physician remembers to follow Fleming's four General Rules and to use sensitivity cultures, his therapeutic failures will decrease to the point that he will not be eager to follow the mirage of polyantibiotic therapy.

In my opinion, one of the more unfortunate practices has been the injection of penicillin and streptomycin in fixed ratios. If one uses these ready-made mixtures, he either gives too little penicillin to be effective, or, if he gives enough penicillin, the dose of streptomycin will be so large as to risk serious permanent damage to the eighth nerve structures.

In the early days of penicillin therapy both patients and doctors accepted the idea of intramuscular injections without protest because of the spectacularly good results. Times have changed. With the advent of the "broad-spectrum" antibiotics, patients learned that orally administered antibiotics could often be effective, and the reports of a few fatal anaphylactic reactions to penicillin given by injection aroused such a phobia in the minds of many physicians that they gave up this mode of administration entirely. In my experience, the phobia about severe penicillin anaphylactic reactions is unwarranted. They are extremely rare, and can be obviated if one inquires about previous adverse reaction to administration of penicillin and if use of penicillin by injection is avoided in known asthmatic patients.

The comparative merits of the various antibiotics can be determined rather accurately by clinical evaluation of their effect in treatment of acute otitis media, because a reliable sample of exudate can be obtained for sensitivity cultures and because the endpoint of the infection can be accurately determined by daily observation of the drum, especially if one uses a pneumatic otoscope as he should.

The fact that penicillin is superior to the sulfonamide drugs was well demonstrated by Wilcox.[11] In 42% of his cases use of sulfadiazine was discontinued because of failure to cure the infection or because of sulfonamide intolerance. Sulfonamide

idiosyncrasy also occurs with the newer preparations.

My results [12] with the use of fortified procaine penicillin have been so spectacularly good in the treatment of otitis media and early mastoiditis that I continue to use it for many patients who have no history of penicillin allergy.

With the ever-increasing number of trade names for the same antibiotic, there will be far less confusion and misunderstanding if we use the generic or official names of the drugs we prescribe.

The past year I have been advising tetracycline by mouth for early infections, but if the infection does not show prompt response within 48 hours, I use fortified procaine penicillin by intramuscular injection with 1,000,000 units five times a day as the average dose for an adult. Finland and associates [13] showed that tetracycline has essentially the same antibacterial activity as oxytetracycline and chlortetracycline, and produced a distinctly lower incidence of gastrointestinal symptoms. Therefore I think it is the drug of choice if one decides to use a drug of the tetracycline group. The dose utilized for most adults is 500 mg. four times daily.

Judging from recently published articles, tetracycline phosphate complex should be more effective than tetracycline hydrochloride, but I have not had enough clinical experience with the phosphate complex to verify these statements.

When the organisms are sensitive to erythromycin but not to tetracycline, I use erythromycin in the same dose, but find that there is a higher incidence of gastrointestinal symptoms.

Novobiocin is a good antistaphylococcal agent, but Martin and associates [14] report that it produces some type of allergic dermatitis in about 9% of the patients. For infections due to penicillin-resistant staphylococci, we use 500 mg. of novobiocin every six hours.

We use chloramphenicol only when the sensitivity cultures indicate the organisms are not sensitive to one or more of the other antibiotics, because of the few reported cases of aplastic anemia associated with the use of chloramphenicol. Welch [15] points out that there are relatively few cases in which aplastic anemia was associated with the use of chloramphenicol, and that most of these patients were children under 10 years of age.

Penicillin V (phenoxymethyl penicillin) has been shown to produce higher blood levels than comparable doses of penicillin G when orally administered, so it is logical to use penicillin V if one elects to use oral penicillin therapy.

Martin and associates [16] have shown that for infections of moderate severity in soft tissues 400,000 units of penicillin V administered every four hours can be a satisfactory method of treatment. Gastrointestinal irritation of moderate degree occurs in about 5% of the patients treated with penicillin V.

My experience indicates that there is something about infections in bone-surrounded cavities that makes them respond poorly and slowly to average doses of penicillin. For this reason, when using penicillin V, I advise 1,000,000 units every four hours.

Despite all that has been written about the possibility of serious anaphylactic reactions to penicillin given by injection, whenever patients with middle ear or mastoid infection fail to show prompt response to orally administered antibiotics, we admit them to the hospital and give no less than 1,000,000 units of fortified procaine penicillin intramuscularly every eight hours. Because of insistence on this program, no patient who has come to us for initial treatment of the ear infection has required a mastoid operation during the past 12 years. Myringotomy is of course always employed. Myringotomy may need to be repeated two, three, or four times because when patients are receiving antibiotic therapy the initial incision may close before exudate has ceased to form in the middle ear.

Table 2, showing the incidence of "simple" mastoid operations at the Geisinger Hospital during the last 20 years, presents some interesting data, not entirely comfort-

TABLE 2.—*Incidence of "Simple" Mastoid Operations at the Geisinger Hospital*

| Year | Period | Cases of Acute Otitis Media and Mastoiditis, No. | "Simple" Mastoid Operations, No. | Percentage Requiring Mastoidectomy |
|---|---|---|---|---|
| 1937 | Presulfonamide | 202 | 119 | 58.9 |
| 1943 | Sulfonamide | 282 | 74 | 26.2 |
| 1946 | Small dose penicillin | 247 | 10 | 4.0 |
| 1949 | Large dose penicillin | 189 | 5 | 2.7 |
| 1950 | (1,000,000-3,000,000 units daily) | 158 | 9 | 5.7 |
| 1951 | (1,000,000-3,000,000 units daily) | 109 | 1 | 0.9 |
| 1952 | (1,000,000-3,000,000 units daily) | 110 | 3 | 2.7 |
| 1953 | Larger dose penicillin | 122 | 0 | 0 |
| 1954 | (5,000,000-10,000,000 units daily) | 65 | 2 | 3.0 |
| 1955 | (5,000,000-10,000,000 units daily) | 106 | 4 | 3.8 |
| 1956 | (5,000,000-10,000,000 units daily) | 69 | 5 | 7.2 |

ing. Please note that the percentage of patients requiring mastoidectory has risen steadily during the past three years. With reference to the probability of preventing a mastoid operation, the patient with acute otitis media under the care of the average practitioner is worse off now than he was in 1946 in the early days of penicillin therapy. In an attempt to determine why, I have analyzed the charts of the nine patients who required "simple" mastoid operations during 1955 and 1956.

The reasons can be summarized as follows:

1. Misplaced confidence in the effectiveness of glycerine ear drops
2. Failure to employ early myringotomy
3. Delay in use of antibiotic therapy until bone necrosis has occurred
4. Inadequate amounts of antibiotic
5. Organism not sensitive to the drug used

Each patient had one or more of the following indications for mastoidectomy:

1. Persistent discharge from the ear (nine cases)
2. Continued pain and fever (seven cases)
3. Vertigo (one case)
4. Facial paralysis (three cases)
5. Large subperiosteal abscess (three cases)

## Summary and Conclusions

The principles of effective antibiotic therapy were outlined by Alexander Fleming in 1946.

These principles apply to use of antibiotics developed since that time.

Presently available antibiotics are effective for nearly all otolaryngologic infections if they are used early, in adequate dose, and with the guidance of sensitivity cultures.

Antibiotic mixtures are seldom necessary if the principles noted above are followed.

Use of antibiotics is no substitute for surgery when there is a localized collection of pus.

Foss Clinic.

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# Additional Variables on the Békésy-Type Audiometer

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## Introduction

The development of the automatic audiometer reported by Békésy[2] in 1947 has provided the clinician with an extremely useful diagnostic instrument. However, despite the need for the early investigation of the various factors in the instrument-patient combination,[8] published research on this type of audiometry has been extremely limited. The first systematic study on the Békésy-type audiometer, in which the effects of testing time and signal attenuation rate were investigated, was reported by Corso[4] in 1955. It was found that for inexperienced listeners (1) testing time and attenuation rate had no effect on the mean threshold value at 1000 cps; (2) attenuation rate produced a significant effect on threshold variability, and (3) attenuation rate had a significant effect on test-retest reliability. In a later study, Corso[5] made a comprehensive comparison of threshold data obtained by the method of limits and the method of adjustment. In the analysis of data, a new and more valid technique for the reading of the Békésy-type audiogram was devised. The results of the study indicated that, with the new technique, there is considerable agreement between the mean threshold values obtained on a Békésy-type audiometer and those obtained by a conventional discrete-frequency audiometer.

The present study was undertaken to extend the evaluation of the Békésy-type audiometer by investigating additional variables which might be utilized in operating the instrument in a clinical situation. Specifically, the primary objective was to study the effects and interactions of four variables on the absolute threshold of hearing: (1) testing time, (2) signal attenuation rate, (3) direction of frequency sweep, and (4) tone-pulsing. The variables of testing time and attenuation rates were included to supplement the data previously reported.[4] A secondary objective of the study was to determine the test-retest reliability of threshold measures obtained under each of the experimental conditions of the study.

## Apparatus

The apparatus consisted of a Békésy-type audiometer, Grason-Stadler model, Type E-524. Basically, the unit is the same type as that described in detail by Reger.[8] The signal output from the General Radio beat-frequency oscillator, Type 1304-A, was presented monaurally to the subject through the right unit of a set of Permoflux PDR-10 earphones set in MX-41/AR cushions. The left unit was used for intercommunication between the tester and the subject. The frequency range covered in threshold testing was 5.5 octaves extending from 200 to 10,000 cps. The testing times investigated were 2.7 and 11.0 minutes; the rates of signal attenuation were 1.5 and 5.0 db/sec. For the pulsed-tone condition, the auditory signal was presented in cycles of 1 second

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on and 1 second off by means of the electronic switch provided in the Grason-Stadler audiometer.

The general procedure in the calibration of the audiometer followed that previously reported by Corso.[6] Calibration data were obtained on (1) frequency-dial markings on the General Radio oscillator, (2) step characteristics of the hearing-loss attenuator, (3) frequency and attenuation markings on the audiogram blank, and (4) sound-pressure output of the earphones into a National Bureau of Standards Coupler 9-A.[7]

## Procedure

### Experimental Design

The subjects in this study were 10 volunteers (5 male, 5 female) from an introductory course in psychology at the Pennsylvania State University. All subjects were between the ages of 17 and 25 years, inclusive. Each subject served as his own control, i. e., each subject was tested under all experimental conditions. The subjects were individually tested in an anechoic chamber which was provided with a two-way voice communication system and a closed-loop television circuit.

Since there were four variables under investigation (testing time, attenuation rate, direction of frequency sweep, and tone-pulsing) with each subject serving as his own control, a matched-group, four-factor analysis of variance experimental design was used.[8] In the present study, there were 16 experimental conditions obtained by combining two levels of each of the four variables: (1) 2.7 and 11.0 minutes testing time, (2) 1.5 and 5.0 db/sec. attenuation rate, (3) low-high and high-low direction of frequency sweep, and (4) pulsed and continuous type of stimulus tone.

The conditions of the study were administered to each subject in a random order. When these tests were completed, the same conditions were again administered to each subject in a random order to obtain a measure of test-retest reliability. No testing session lasted for more than one hour, and no subject served for more than one session per day. The design of the present study, in which the same subjects were tested under each experimental condition, provided greater precision than would have been possible by testing 160 subjects, with 10 subjects randomly assigned to each of the 16 conditions.[9]

### Testing Method

The general testing method was the same for all experimental conditions. After the subject was comfortably seated in the testing room, the tester read aloud an appropriate set of instructions. He then removed any eyeglasses worn by the subject and, for female subjects, pushed the hair away from the ears. The earphones were next positioned over the ears with sufficient tension in the headband spring to hold the earphones firmly in place throughout the test. The subject was informed by means of the intercommunication system whenever the test was about to begin.

Each test was started at 200 cps at a sound pressure level of 45 db. re 0.0002 dyne/cm.$^2$ For the experimental conditions involving a high-low direction of frequency sweep, the test tone was presented at the same sound pressure level, but at 10,000 cps. Threshold measurements were made on right ears only.

The specific set of instructions for each experimental condition followed the same general pattern; but, in each case, the instructions were appropriately modified for the given condition. An example of these instructions is presented for a condition involving a continuous stimulus tone and a low-high direction of frequency sweep.

"I am going to run a test to find out how 'soft' a tone you can hear in your right ear. The tone you will hear will start out very 'low'; that is, it will sound something like a low note on the piano. The tone will then get gradually higher and higher in pitch. Your task is to adjust the loudness of this tone so that it is always just barely audible. If you want the tone to get 'softer' or 'fade away,' just press this switch and hold it down. This will cause the tone, to get so 'soft' that you will no longer be able to hear it. So, in order to get the tone louder again, release the switch. The tone will then gradually 'build up.' You must listen very carefully, pressing and releasing the switch in such a way that the tone is always just barely audible. Remember, to make the tone get 'softer,' press the switch; to make the tone get louder, release the switch. This sign (tester points to sign) will remind you of these directions. Are there any questions? [Pause.] After I have placed the earphones on your head, do not touch them in any way. If you should need to talk to me for any reason, just speak up and I will hear you over this microphone."

## Results and Comment

The threshold values for the various conditions of the present study were obtained by reading the "peak" values of the Békésy-type audiograms as described by Corso.[5] To minimize any practice effects resulting from each subject's being tested under all conditions, retest audiograms were used in the evaluation of the factors under study. It was assumed that after 16 different audiometric tests, further practice (retests)

TABLE 1.—*Mean Retest Threshold Values in Decibels re 0.0002 Dyne/Cm.² at 1000 CPS*

| | Continuous Tone | | | | | | | | Pulsed Tone | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Low-High Direction of Frequency Sweep | | | | High-Low Direction of Frequency Sweep | | | | Low-High Direction of Frequency Sweep | | | | High-Low Direction of Frequency Sweep | | | |
| Testing time (Min.): | 2.7 | | 11.0 | | 2.7 | | 11.0 | | 2.7 | | 11.0 | | 2.7 | | 11.0 | |
| Attenuation rate (db/sec.): | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 |
| Frequency in cps | | | | | | | | | | | | | | | | |
| 300 | 31.7 | 23.4 | 28.4 | 24.8 | 22.5 | 18.1 | 25.6 | 19.7 | 37.9 | 24.8 | 28.5 | 24.2 | 25.7 | 16.5 | 26.5 | 20.9 |
| 500 | 18.7 | 12.0 | 18.4 | 18.0 | 13.3 | 10.1 | 17.6 | 14.6 | 22.0 | 13.3 | 22.2 | 14.4 | 17.1 | 8.3 | 19.1 | 12.0 |
| 750 | 10.7 | 6.0 | 11.1 | 10.0 | 8.8 | 5.1 | 13.7 | 18.0 | 14.2 | 20.2 | 13.6 | 8.8 | 12.3 | 4.3 | 13.1 | 9.3 |
| 1000 | 11.1 | 0.3 | 8.3 | 5.0 | 4.8 | −0.1 | 12.2 | 8.9 | 11.1 | 0.3 | 8.4 | 14.8 | 9.2 | 1.3 | 11.2 | 6.4 |
| 1500 | 5.1 | −4.0 | 7.5 | 3.9 | 6.9 | 2.9 | 10.1 | 8.5 | 9.3 | 0.9 | 8.7 | 1.5 | 10.3 | 1.9 | 11.2 | 8.9 |
| 2000 | 5.9 | −2.5 | 11.4 | 7.2 | 10.5 | 8.6 | 14.6 | 9.7 | 7.9 | 2.9 | 11.2 | 6.1 | 9.8 | 3.5 | 10.0 | 10.4 |
| 3000 | 4.8 | −1.9 | 10.8 | 8.4 | 11.6 | 3.8 | 14.4 | 7.5 | 7.9 | 5.5 | 6.5 | 0.1 | 9.8 | 4.5 | 7.6 | 7.2 |
| 4000 | 3.1 | −3.7 | 6.7 | 4.5 | 16.0 | 9.5 | 14.1 | 10.8 | 6.2 | −0.5 | 4.1 | 0.0 | 15.4 | 3.5 | 8.8 | 3.2 |
| 6000 | 8.9 | 2.0 | 14.7 | 18.2 | 29.2 | 13.5 | 19.2 | 15.9 | 10.2 | 5.9 | 13.4 | 8.4 | 23.4 | 10.2 | 17.5 | 10.4 |
| 7000 | 9.7 | −0.5 | 11.9 | 14.5 | 32.4 | 9.8 | 20.0 | 12.4 | 10.8 | 6.0 | 12.0 | 8.8 | 25.1 | 7.1 | 15.5 | 7.1 |

would have little effect on threshold values. The mean retest threshold values in db. re 0.0002 dyne/cm.² for the 10 subjects of this study are given in Table 1 for 10 different frequencies from 300 to 7000 cps, inclusive.

The statistical treatment of data to determine the significance of the effects of the four variables on the threshold of hearing involved a conventional analysis of variance[6] on the absolute threshold values at 1000 cps. The results of this analysis are shown in Table 2. Three sources of variance produced *F*-ratios significant at the 1% level: attenuation rate, testing time, and individuals. Since the mean threshold value at 1000 cps for all conditions involving a 1.5 db/sec. attenuation rate was 8.9 db., compared with a mean value of 3.5 db. for all conditions involving a 5.0 db/sec. rate, it appears that increasing the attenuation rate from 1.5 to 5.0 db/sec. results in a lower threshold. However, increasing the testing time from 2.7 to 11.0 minutes raises the mean threshold value from 3.9 to 7.9 db. re 0.0002 dyne/cm.². The significant *F*-ratio for individuals shows that, as expected, there are real differences in hearing sensitivity at 1000 cps. The direction of frequency sweep and the type of stimulus tone (continuous vs. pulsed) failed to affect the 1000 cps threshold value. All interactions of the four variables failed to attain statistical significance.

The finding that the variables of attenuation rate and testing time significantly affect the mean threshold value at 1000 cps appears inconsistent with the data previously reported by Corso.[4] However, when the

TABLE 2.—*Analysis of Variance of Threshold Values at 1000 CPS*

| Source of Variance | Sum of Squares | Degrees of Freedom | Mean Square | *F*-Ratio |
|---|---|---|---|---|
| Main Effects | | | | |
| Attenuation rate | 1467.13 | 1 | 1467.13 | 55.57 * |
| Testing time | 644.41 | 1 | 644.41 | 24.41 * |
| Direction of frequency sweep | 60.40 | 1 | 60.40 | 2.29 |
| Type of stimulus tone | 75.49 | 1 | 75.49 | 2.86 |
| First order interactions | | | | |
| Rate×time | 25.14 | 1 | 25.14 | |
| Rate×direction | 9.36 | 1 | 9.36 | |
| Rate×type | 11.29 | 1 | 11.29 | |
| Time×direction | 88.06 | 1 | 88.06 | 3.34 |
| Time×type | 91.36 | 1 | 91.36 | 3.46 |
| Direction×type | 0.14 | 1 | 0.14 | |
| Second order interactions | | | | |
| Rate×time×direction | 57.24 | 1 | 57.24 | 2.17 |
| Rate×time×type | 38.32 | 1 | 38.32 | 1.45 |
| Rate×direction×type | 1.66 | 1 | 1.66 | |
| Time×direction×type | 0.04 | 1 | 0.04 | |
| Third order interaction | | | | |
| Rate×time×direction×type | 2.79 | 1 | 2.79 | |
| Individuals | 7016.52 | 9 | 779.61 | 29.53 * |
| Residual | 3564.19 | 135 | | |
| Total | 13153.43 | 159 | | |

* $p<0.01$.

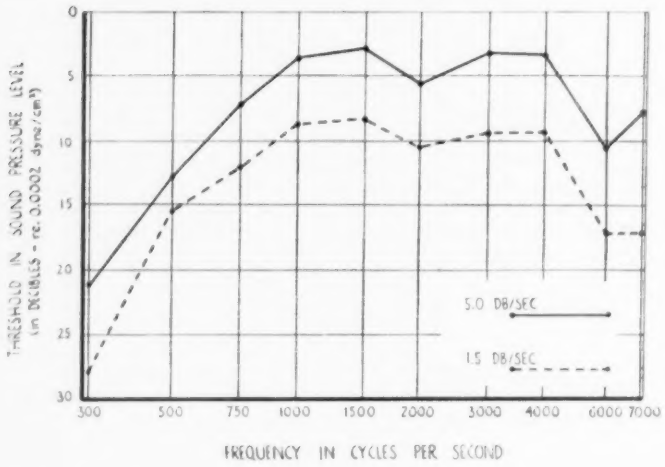


Fig. 1.—Mean threshold curves obtained with different attenuation rates.

Békésy audiograms used in the Corso[1] study were reread to provide "peak" values rather than the reported midpoint values, the data agreed very well. In the Corso[1] study, for a 1 db/sec. attenuation rate and a 10 minute testing time, the mean retest threshold value for 10 inexperienced subjects was 12.2 db.; in the present study, for a 1.5 db/sec. rate and an 11.0 minute testing time, the mean retest threshold value for 10 experienced subjects was 8.3 db. It is conceivable that the 4 db., approximately, lower mean threshold value obtained in the present study reflects the effects of practice in the testing situation.

Figure 1 presents a comparison of the mean threshold curve obtained from the eight conditions involving a 5.0 db/sec. attenuation rate and the mean threshold curve obtained from the other eight conditions involving a 1.5 db/sec. rate. Each curve is based on the mean retest threshold values in db. re 0.0002 dyne/cm.$^2$ at the following frequencies: 300, 500, 750, 1000, 1500, 2000, 3000, 4000, 6000, and 7000 cps. Inspection of Figure 1 shows that the 5.0 db/sec. attenuation rate yields lower mean threshold values throughout the frequency range tested.

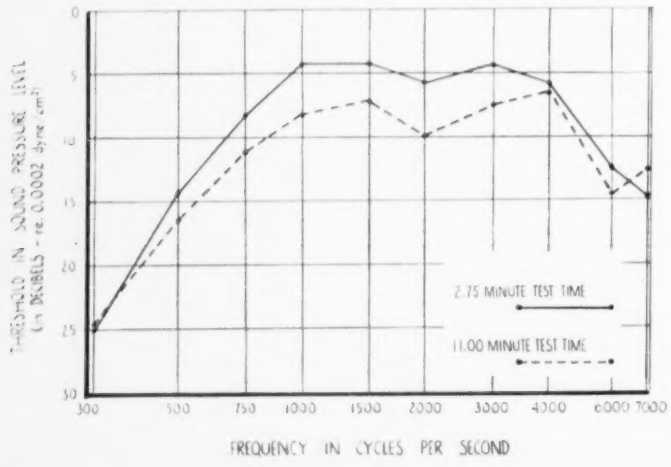


Fig. 2.—Mean threshold curves obtained with different testing times.

TABLE 3.—*Analysis of Variance of Threshold Values at 300 CPS*

| Source of Variance | Sum of Squares | Degrees of Freedom | Mean Square | *F*-Ratio |
|---|---|---|---|---|
| Main effects | | | | |
| Attenuation rate | 1739.76 | 1 | 1739.76 | 58.88 * |
| Testing time | 0.10 | 1 | 0.10 | |
| Direction of frequency sweep | 1422.06 | 1 | 1422.06 | 48.12 * |
| Type of stimulus tone | 74.53 | 1 | 74.53 | 2.52 |
| First order interactions | | | | |
| Rate×time | 130.32 | 1 | 130.32 | 4.41 † |
| Rate×direction | 10.92 | 1 | 10.92 | |
| Rate×type | 60.03 | 1 | 60.03 | 2.02 |
| Time×direction | 308.58 | 1 | 308.58 | 10.44 * |
| Time×type | 40.00 | 1 | 40.00 | 1.35 |
| Direction×type | 4.16 | 1 | 4.16 | |
| Second order interactions | | | | |
| Rate×time×direction | 78.12 | 1 | 78.12 | 2.64 |
| Rate×time×type | 59.54 | 1 | 59.54 | 2.01 |
| Rate×direction×type | 0.00 | 1 | 0.00 | |
| Time×direction×type | 32.58 | 1 | 32.58 | 1.10 |
| Third order interaction | | | | |
| Rate×time×direction×type | 2.76 | 1 | 2.76 | |
| Individuals | 8150.04 | 9 | 905.56 | 30.64 * |
| Residual | 3842.02 | 130 ‡ | 29.55 | |
| Total | 15955.52 | 154 | | |

* $p \leq 0.01$.
† $p \leq 0.05$.
‡ 5 df lost in estimating missing values.

Figure 2 presents a comparison of the mean threshold curve obtained for the eight conditions involving a 2.7 minute testing time and the mean threshold curve obtained for the other eight conditions involving an 11.0 minute testing time. Each curve is based on the same 10 test frequencies as used in Figure 1. Inspection of Figure 2 shows that the 2.7 minute testing time yields lower mean threshold values for all frequencies except 300 and 7000 cps, with the most marked differences occurring between 1000 and 3000 cps.

To determine the statistical significance of the direction of frequency sweep variable, an analysis of variance was performed on the threshold values obtained at both 300 and 7000 cps. In the analyses, it was necessary to estimate five missing threshold values at each of these frequencies. The technique used in these estimates was that devised by Yates.[3] The results of the analysis for 300 cps are presented in Table 3.

Two main effects are significant at the 1% level: attenuation rate and direction of frequency sweep. In addition, two first-order interactions are significant at or beyond the 5% level: rate by time, and time by direction. As in the 1000 cps case, individual differences are statistically significant at the 1% level.

The mean threshold value at 300 cps obtained for the eight conditions involving a 1.5 db/sec. attenuation rate was 28.3 db.; for the eight conditions involving a 5.0 db/sec. rate, the mean threshold was 21.6 db. Thus, as for 1000 cps, an increase in the attenuation rate produces a significant decrease in the absolute threshold of hearing.

For the eight conditions using a high-low direction of frequency sweep, the mean threshold was 21.9 db.; for the eight conditions involving a low-high direction, the mean threshold was 27.9 db. Apparently, under the conditions of the present study, the thresholds obtained for frequencies in the initial phase of the test tend to be spuriously high when a low-high direction of frequency sweep is used in Békésy-type audiometry.

The conditions in which a 1.5 db/sec. rate was combined with a 2.7 minute testing time and those in which a 5.0 db/sec. rate was combined with an 11.0 minute testing time resulted in a mean threshold of 25.7 db. re 0.0002 dyne/cm.$^2$ On the other hand, when a 5.0 db/sec. rate was combined with a 2.7 minute testing time and a 1.5 db/sec. rate with an 11.0 testing time, a mean threshold of 23.9 db. was obtained. Thus, since the mean threshold of 23.9 closely agrees with Sivian and White's [10] threshold of 22.0 db. at 300 cps, it would seem that a more valid

estimate of threshold is obtained when a fast testing time is combined with a fast rate, or a slow time with a slow rate. Optimal results seem to depend upon some consistency between the rate at which the tone increases or decreases in intensity and the rate at which the frequency range is swept.

The conditions in which a low-high direction of frequency sweep was combined with an 11.0 minute testing time and those in which a high-low direction was combined with a 2.7 minute testing time yielded a mean threshold of 23.4 db. When the low-high direction was combined with a 2.7 minute testing time and a high-low direction with an 11.0 minute testing time, a mean threshold of 26.2 db. was obtained. Again, on the basis of the Sivian and White [10] data at 300 cps, the most valid estimate of threshold at 300 cps is obtained when the criterion threshold (300 cps) is presented either at the beginning of an 11.0 minute test, or at the end of a 2.7 minute test.

Table 4 presents the results of the analysis of variance performed on the threshold values at 7000 cps. Three main effects are significant at or beyond the 5% level: attenuation rate, direction of frequency sweep, type of stimulus tone; three first order interactions are significant at the 1% level: rate by time, rate by direction, and time by direction; individual differences are again significant at the 1% level.

For those conditions involving a 1.5 db/sec. attenuation rate, the obtained mean threshold at 7000 cps was 17.3 db., while those conditions with a 5.0 db/sec. rate yielded a mean threshold of 8.4 db. These results again showed a consistent effect that a faster attenuation rate produces a lower mean threshold value.

For those conditions involving a low-high direction of frequency sweep, the obtained mean threshold at 7000 cps was 9.4 db., for the conditions involving a high-low direction, the mean threshold was 14.4 db. As in the 300 cps case, the lower mean threshold value is obtained when the criterion frequency is presented near the end of the test rather than in the beginning.

Figure 3 presents a comparison of the mean threshold values for the low-high and high-low directions of frequency sweep for 10 test frequencies (300, 500, 750, 1000, 1500, 2000, 3000, 4000, 6000, and 7000 cps). All threshold values are based on retest audiograms and are expressed in db. re 0.0002 dyne/cm.$^2$ Inspection of the curves shows that a lower threshold value is obtained for a given frequency when that frequency occurs toward the end of a given test. The two curves cross at approximately

TABLE 4.—*Analysis of Variance of Threshold Values at 7000 CPS*

| Source of Variance | Sum of Squares | Degrees of Freedom | Mean Square | *F*-Ratio |
|---|---|---|---|---|
| Main effects | | | | |
| Attenuation rate | 3203.21 | 1 | 3203.21 | 65.90* |
| Testing time | 5.44 | 1 | 5.44 | |
| Direction of frequency sweep | 2007.18 | 1 | 2007.18 | 41.29* |
| Type of stimulus tone | 311.64 | 1 | 311.64 | 6.41† |
| First order interactions | | | | |
| Rate×time | 901.07 | 1 | 901.07 | 18.54* |
| Rate×direction | 1149.72 | 1 | 1149.72 | 23.65* |
| Rate×type | 1.43 | 1 | 1.43 | |
| Time×direction | 1109.33 | 1 | 1109.33 | 22.82* |
| Time×type | 41.72 | 1 | 41.72 | |
| Direction×type | 284.89 | 1 | 284.89 | 5.86 |
| Second order interactions | | | | |
| Rate×time×direction | 84.53 | 1 | 84.53 | 1.74 |
| Rate×time×type | 137.83 | 1 | 137.83 | 2.84 |
| Rate×direction×type | 35.81 | 1 | 35.81 | |
| Time×direction×type | 125.49 | 1 | 125.49 | |
| Third order interaction | | | | |
| Rate×time×direction×type | 8.15 | 1 | 8.15 | |
| Individuals | 6985.40 | 9 | 776.16 | 17.96* |
| Residual | 6320.50 | 130‡ | 48.61 | |
| Total | 22713.34 | 154 | | |

* $p<0.01$.
† $p<0.05$.
‡ 5 df lost in estimating missing values.

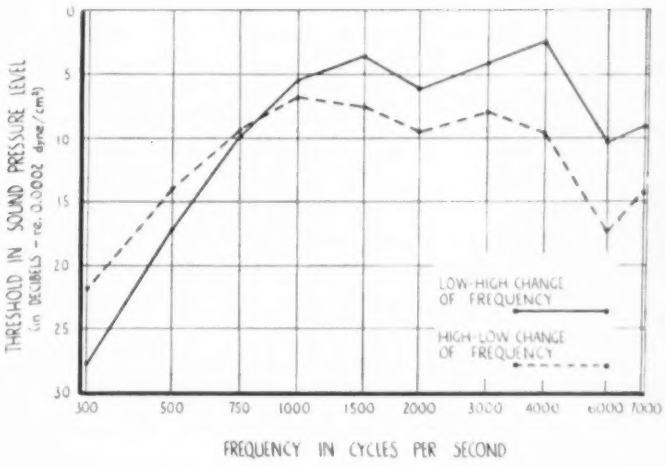


Fig. 3.—Mean threshold curves obtained with two directions of frequency sweep.

750 cps, indicating that in this region the direction of frequency sweep has no effect.

For those conditions involving a continuous type of stimulus tone, the obtained mean threshold at 7000 cps was 14.2 db.; for those conditions with a pulsed type of tone, the mean threshold was 11.4 db. Thus, tone-pulsing tended to lower the threshold value by 2.8 db. at 7000 cps.

Figure 4 presents a comparison of mean threshold values for the continuous and pulsed types of stimulus tone for the same test frequencies as shown in Figure 3. For frequencies below 2000 cps, tone-pulsing appears to increase the threshold value, but these differences are not statistically significant as indicated in the analyses for 1000 cps and 300 cps. Above 2000 cps, tone-pulsing appears to lower significantly the mean threshold value, with the largest differences occurring in the 3000-4000 cps region. These findings are consistent with those of Rosenblith and Miller,[9] who reported that, in the method of limits, the difference in threshold between continuous and interrupted tones increases as the frequency of the tone is increased, with the maximum difference at about 4000 cps.

As in the 300 cps analysis, the interaction of attenuation rate and testing time produced

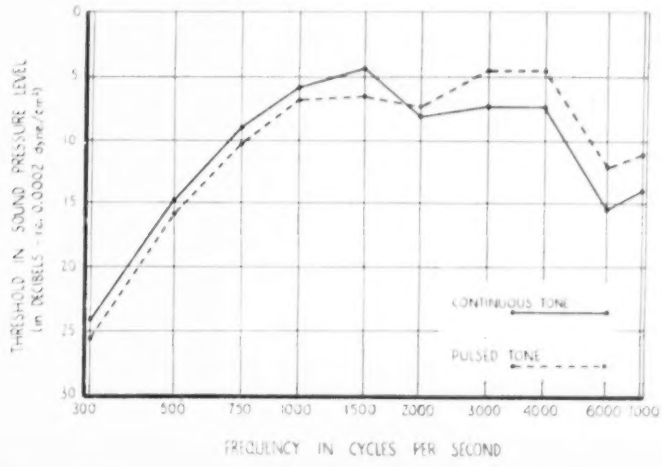


Fig. 4.—Mean threshold curves obtained with two types of stimulus tones.

TABLE 5.—*Median Test-Retest Reliability Coefficients*

| | Continuous Tone | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| Direction of frequency sweep | Low-High | | | | High-Low | | | |
| Testing time (min.) | 2.7 | | 11.0 | | 2.7 | | 11.0 | |
| Attenuation rate (db/sec.) | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 |
| Median reliability coefficient | 0.92 | 0.77 | 0.79 | 0.60 | 0.94 | 0.64 | 0.72 | 0.60 |
| | Pulsed Tone | | | | | | | |
| Direction of frequency sweep | Low-High | | | | High-Low | | | |
| Testing time (min.) | 2.7 | | 11.0 | | 2.7 | | 11.0 | |
| Attenuation rate (db/sec.) | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 | 1.5 | 5.0 |
| Median reliability coefficient | 0.90 | 0.82 | 0.88 | 0.75 | 0.84 | 0.77 | 0.84 | 0.62 |

a significant effect on the 7000 cps threshold. The smallest threshold value was obtained when there was some consistency between attenuation rate and testing time. Those conditions involving a combination of 1.5 db/sec. rate with a 2.7 minute time, or a 5.0 db/sec. rate with an 11.0 minute time, yielded a mean threshold value of 15.2 db. With a 5.0 db/sec. rate and a 2.7 minute time or a 1.5 db/sec. rate and an 11.0 minute time, the mean value was 10.5 db. Again, a slow rate and a slow time or a fast rate and a fast time produce a significantly lower mean threshold value.

For the interaction of attenuation rate and frequency direction, a combination of 1.5 db/sec. rate with a low-high direction of frequency sweep and a combination of 5.0 db/sec. rate with a high-low direction of sweep resulted in a mean threshold of 10.2 db. at 7000 cps. On the other hand, a 5.0 db/sec. rate combined with a low-high direction and 1.5 db/sec. rate with a high-low direction of sweep yielded a mean threshold of 15.5 db. The latter combination of conditions represents a significant increase of 5.3 db. in the threshold value at 7000 cps.

As in the 300 cps analysis, the interaction of testing time and direction of frequency change produced a significant change in the mean threshold value at 7000 cps. A 2.7 minute testing time with a low-high direction of frequency sweep and an 11.0 minute testing time with a high-low direction yielded a mean threshold of 10.2 db. This value was 5.3 db. lower than the mean threshold for the same frequency when an 11.0 minute testing time and a low-high direction or a 2.7 minute time with a high-low direction of sweep were used.

TABLE 6.—*Analysis of Variance of Z-Transformed Reliability Coefficients*

| Source of Variance | Sum of Squares | Degrees of Freedom | Mean Square | *F*-Ratio |
|---|---|---|---|---|
| Main effects | | | | |
| Attenuation rate | 9.312 | 1 | 9.312 | 45.87* |
| Testing time | 3.184 | 1 | 3.184 | 15.68* |
| Direction of frequency sweep | 1.363 | 1 | 1.363 | 6.71* |
| Type of stimulus tone | 0.179 | 1 | 0.179 | |
| First order interactions | | | | |
| Rate×time | 0.064 | 1 | 0.064 | |
| Rate×direction | 0.148 | 1 | 0.148 | |
| Rate×type | 0.327 | 1 | 0.327 | 1.61 |
| Time×direction | 0.046 | 1 | 0.046 | |
| Time×type | 0.086 | 1 | 0.086 | |
| Direction×type | 0.776 | 1 | 0.776 | 3.82 |
| Second order interactions | | | | |
| Rate×time×direction | 0.081 | 1 | 0.081 | |
| Rate×time×type | 0.485 | 1 | 0.485 | 2.39 |
| Rate×direction×type | 0.091 | 1 | 0.091 | |
| Time×direction×type | 0.005 | 1 | 0.005 | |
| Third order interaction | | | | |
| Rate×time×direction×type | 0.431 | 1 | 0.431 | 2.12 |
| Individuals | 4.180 | 9 | 0.464 | 2.28 |
| Residual | 23.693 | 125† | 0.203 | |
| Total | 44.451 | 149 | | |

* $p<0.01$.
† 10 df lost in estimating missing values.

In addition to the analyses of variance performed on the threshold data at 300, 1000, and 7000 cps, a Pearson product-moment reliability coefficient was computed for each of the experimental conditions. These coefficients were based on 10 arbitrarily selected frequencies from the test and retest audiograms: 300, 500, 750, 1000, 1500, 2000, 3000, 4000, 6000, and 7000 cps. Table 5 presents the median reliability coefficient obtained for each experimental condition of the present study.

To test the effects of the four variables on the reliability of threshold measurements, the correlation coefficient ($r$) for each subject was transformed to a $z$-score, and an analysis of variance was performed on the transformed scores. A summary of the results of this analysis is presented in Table 6. Three main effects reached the 1% level of significance: attenuation rate, testing time, and direction of frequency sweep. The conditions which produced the higher mean test-retest reliability coefficients ($z$-transformations) were the 1.5 db/sec. attenuation rate, the 2.7 minute testing time, and the low-high direction of frequency sweep as compared with the 5.0 db/sec. rate, the 11.0 minute time, and the high-low direction of sweep, respectively. The finding that attenuation rate significantly affects test-retest reliability supports the earlier work of Corso.[4]

## Summary and Conclusions

This study was conducted to determine the effects of four operating conditions on a Békésy-type audiometer on the absolute threshold of hearing: (1) attenuation rate, (2) testing time, (3) direction of frequency sweep, and (4) type of stimulus tone.

A Grason-Stadler model of the Békésy-type audiometer was used to administer hearing tests to 10 volunteer subjects from an introductory course in psychology. All subjects were between 17 and 25 years of age, inclusive. Only right ears were tested, and all auditory measurements were made with the subjects seated in an anechoic chamber. No subject served for more than one hour per testing session, nor for more than one session per day.

A four-factor analysis of variance experimental design was used with each subject being tested and retested under all conditions of the study. Each of the four variables was varied in two ways to provide a total of 16 experimental conditions. The specific operating conditions were (1) 1.5 and 5.0 db/sec. attenuation rate, (2) 2.7 and 11.0 minutes total testing time, (3) low-high and high-low direction of frequency sweep, and (4) continuous and pulsed type of stimulus tone. The obtained absolute thresholds of hearing were expressed in terms of sound-pressure level in db. re 0.0002 dyne/cm.$^2$ as measured in a National Bureau of Standards Coupler 9-A.

To test the significance of the effects of the four variables, an anlysis of variance was performed on the retest threshold values at 1000 cps. Two additional analyses were performed on the retest threshold values at 300 and 7000 cps to test for the significance of the effect of the direction of frequency sweep.

For each subject, a Pearson product moment reliability coefficient was computed for each experimental condition. These coefficients were based on 10 arbitrarily selected frequencies from test and retest audiograms (300, 500, 750, 1000, 1500, 2000, 3000, 4000, 6000, and 7000 cps). To determine the effects of the four variables on the reliability of threshold measurements, all obtained $r$'s were transformed into $z$-scores and examined by an analysis of variance.

The results of this study indicate that the main effects of attenuation rate and testing time on the absolute auditory threshold at 1000 cps are statistically significant beyond the 1% level. The 5.0 db/sec. attentuation rate produces a lower mean threshold throughout the frequency range tested, but the mean thresholds obtained by the 1.5 db/sec. rate are in closer agreement with the Sivian and White[10] values, except at 300, 500, and 750 cps. Also, inspection of

the test-retest reliability coefficients indicates that higher reliability is obtained when the 1.5 db/sec. rate is used.

The 2.7 minute testing time significantly reduced the mean threshold value at 1000 cps, but the 11.0 minute testing time yielded lower thresholds at either end of the frequency range tested. At 1000 cps, the 11.0 minute testing time resulted in a mean threshold essentially identical with that of Sivian and White.[10] However, the 2.7 minute testing time appears to yield the higher test-retest reliability.

The effect of the direction of frequency sweep was found to be significant at the 1% level when the criterion thresholds were taken as 300 and 7000 cps. When the criterion frequency was presented in the initial phase of the test, the obtained threshold was spuriously higher than when it was presented towards the end of the test. The low-high direction of sweep yielded the higher test-retest reliability coefficient.

At 7000 cps, the mean threshold obtained with a pulsed type of stimulus tone was significantly lower than that for the continuous type of tone. However, this effect was not consistent throughout the entire threshold curve. The type of tone had no effect on test-retest reliability.

At both 300 and 7000 cps, the interaction of attenuation rate and testing time and the interaction of testing time and direction of frequency sweep were found to be statistically significant. The best estimates of threshold appear to be obtained when faster rate and faster time are combined and when slower rate and slower time are combined. Also, the interaction of testing time and direction of frequency sweep indicates that the best estimate of threshold at 300 cps, for example, is obtained with a slow testing time and a low-high direction of frequency sweep.

When the validity of the threshold estimates and the test-retest reliability are considered simultaneously, it appears that within the conditions of the present study (including experienced subjects), optimal results on a Békésy-type audiometer are obtained with a 1.5 db/sec. attenuation rate, an 11.0 minute testing time, a low-high direction of frequency change, and either a continuous or a pulsed tone.

Pennsylvania State University.

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# A Simple Method for Calibration of Earphones

**JAMES F. NICKERSON, Ph.D., Bozeman, Mont.**

Calibration of earphones remains a weak link in many operational and research procedures. Often the small laboratory or field installation has difficulty justifying an expensive coupler system for earphone calibration, and even with such systems the process is surprisingly time-consuming.

The problem reported here was twofold—to seek a simple method of calibration which would employ only the more commonly found laboratory equipment and, second, to examine the feasibility of application of such a method to the self-, or internal, calibration of equipment such as the audiometer.

The procedure employed makes use of the fact that an earphone operates both as an earphone and as a microphone, although with markedly differing response characteristics. When an earphone is used as a microphone it is common for the response characteristic to show a decrease in sensitivity below 1000 cps of about 10 db. per octave, and resonance points will shift slightly upward or downward dependent upon the peculiar characteristics of the phone itself and the design of the system of coupling employed.

Thus when an earphone driven by an oscillator is coupled to another earphone for calibration purposes, the response curve will bear little relation to the response curve of the same test phone placed in a standard 6 cc. calibration coupler system. This is particularly true at the lower frequencies. The question of first importance concerns whether these errors between the response of the test phone in the coupler phone and in the 6 cc. standard coupler are consistent for phones of a particular type.

To this end a coupler system employing a Permoflux PDR-8 earphone driven as a microphone was devised. Output from an audio-oscillator, Hewlett Packard Model 200 AB (1 volt, unloaded), was fed through a 300 ohm resistor to each of three Permoflux PDR-8 test phones. Each test phone was held face to face against the PDR-8 coupler phone with a force of 1 kg. cushioned by a cardboard washer 0.040 in. thick with a 0.75 in. circular opening in the center. The output of the coupler phone was given 30 db. of linear amplification and fed to a Ballantine Voltmeter Model 300, and the data of output versus frequency were gathered. The commonly used frequencies from 50 cps to 9000 cps were used, and additional exploration of response at frequencies surrounding apparent points of resonance was made.

The three test phones were then calibrated by means of a JRB 6 cc. coupler with a Western Electric 640 AA microphone, its associated amplifier, and a Ballantine Voltmeter Model 300. Comparisons were made between the 6 cc. coupler calibration data and that of the PDR-8 coupler system by plotting the differences for each phone at each frequency on a single graph. From the line of best fit for the distribution of differences, the necessary correction for each test frequency was determined for the PDR-8 coupler system. In general, the differences were consistent even at the upper frequencies, where earphone response is most erratic. Figure 1 shows the data of differences for the three phones under test. The distribution of differences about the line of

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From the Department of Psychology, Montana State College.

Data collected by the author at U. S. Navy Electronics Laboratory, Human Factors Division, San Diego, Calif.

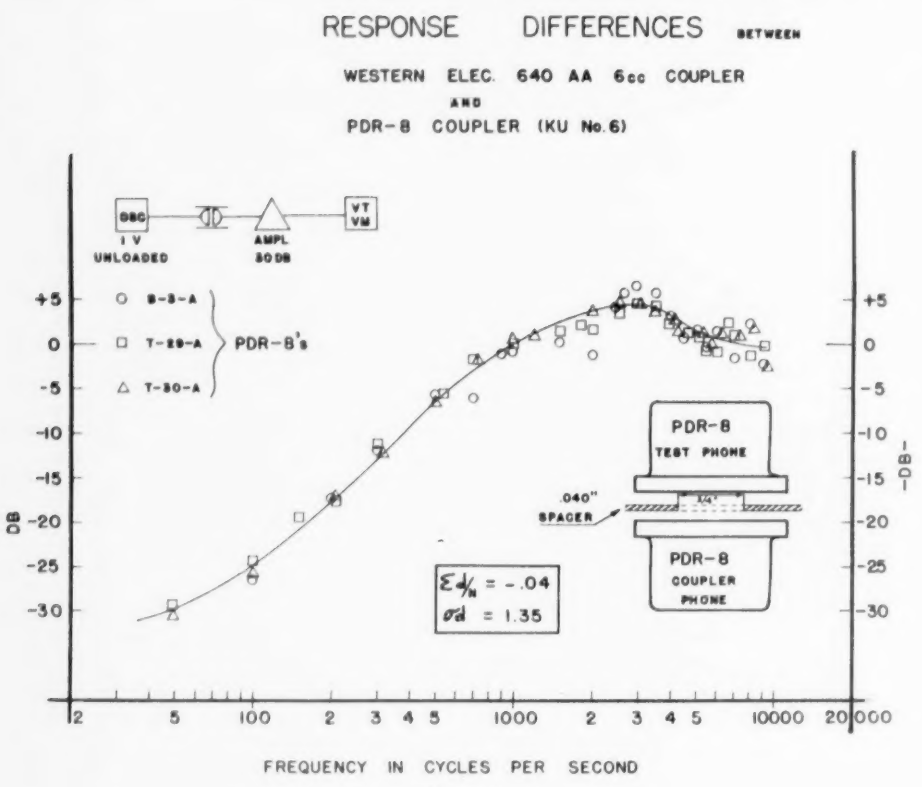


Figure 1

best fit reveals a mean difference of −0.04 db. ($\Sigma d/n = -0.04$) and a standard error of 1.35 db. ($\sigma_d = 1.35$ db.).

Applying the correction data for the PDR-8 coupler system as shown in Figure 1 to the output readings of each of the three test phones gave the calibration curve for each of the phones. Figures 2, 3, and 4 show the calibration of the three phones by the two methods.

Further examination of the distribution of differences showed maximum variation of the three test phones at 700, 1500, and 2000 cps. At these frequencies the line of best fit introduces errors up to 5 db. However, this error could be reduced markedly if the line of best fit followed the median reading per frequency rather than being smoothed to the degree shown in Figure 1 or if the coupler system were applied to calibrating one phone only.

Examining the calibration comparisons closely, it is apparent that there is an excellent correspondence between the two systems, perhaps on the order of calibration effected on a single phone by two different 6 cc. coupler systems. However, there are apparent small shifts in location of resonance peaks and troughs, e. g., Figure 2, 2700 cps and 3000 cps, and Figure 4 at 4600 cps. These differences are largely a function of the individual test phone as well as the particular coupler system.

These observations point up two major limitations that must be placed upon such a method of calibration. First, it appears that the system is apparently optimum when it is used to calibrate a single phone, such as

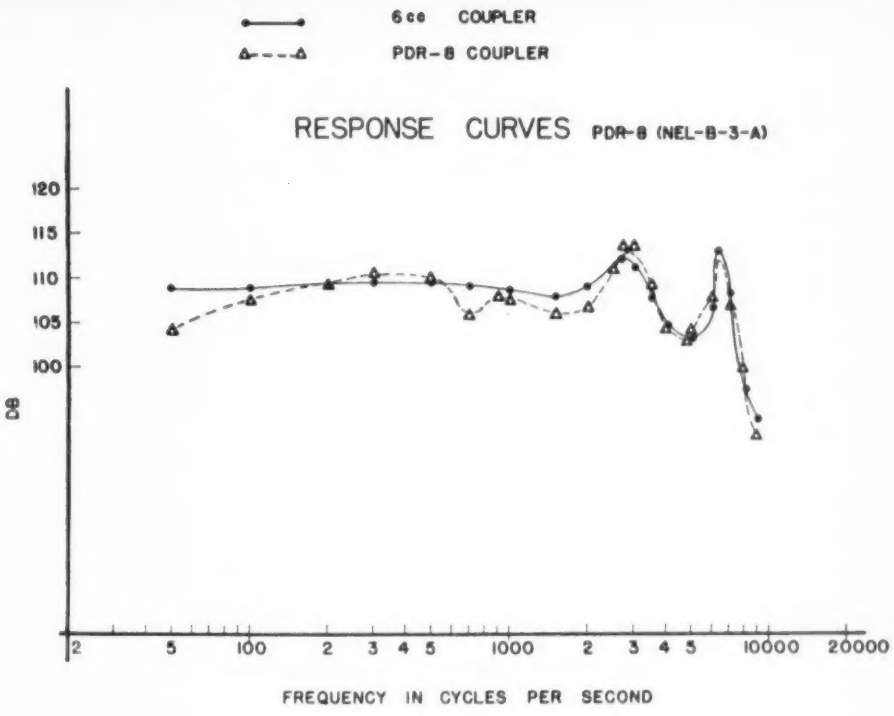


Figure 2

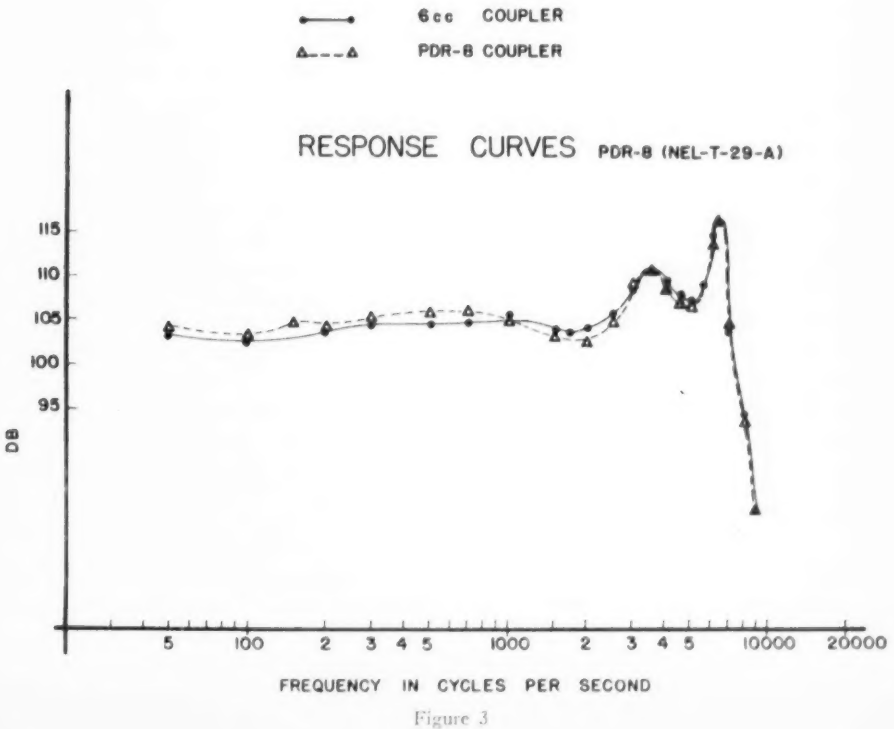


Figure 3

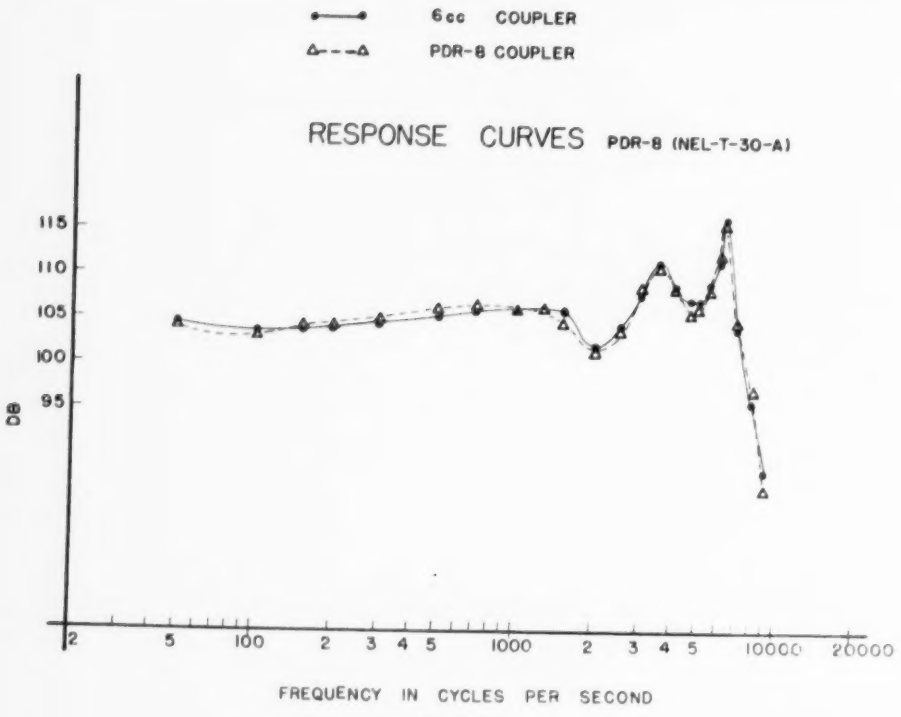


Figure 4

the output phone of an audiometer, for the unique response of a single phone tested under these conditions can be reflected faithfully in the correction curve. However, accuracy still remains at a sufficiently high level in testing phones of a single type (e. g., PDR-8's) by using a fitted correction curve, such as described for the three PDR-8's used above. Presumably the correction curve for one type of phone should not be used for a second type, owing to acoustical impedance differences attributable to the construction of the phone and the differences in volume and shape of the resulting coupling system. It should be possible, however, to effect satisfactory calibration of several differing types of phones, provided that the plot of error and line of best fit is found for each type of phone under test.

A second limitation to bear in mind is that imposed by a piece of equipment such as a pure tone audiometer. Here the precise determination of points of resonance of a phone becomes crucial. It is often necessary to approximate a frequency (say 1000 ± 100 cps) to avoid a resonant point in a given phone. In such cases the use of a coupler phone as a calibrating device must be checked carefully against both the standard coupler and the frequency demands of the audiometer itself. This is not a serious handicap, but where crucial accuracy is necessary additional attention must be given to the response of the system as well as to both the test phone and the coupler phone.

In spite of the limitations suggested above, it appears from these data that it is quite practical and feasible to use a single earphone as a secondary standard for calibration purposes. In practice, it is first necessary to determine the unique response characteristics of the particular phone when used as a coupler phone. Moreover, it will be necessary to determine the response characteristics of this coupler phone for a

single test phone or for each of the several types of phones to be tested. In each case the necessary correction curves can be supplied with the coupler phone for its use as a calibration system.

The many applications of this method are left to the reader, but the application to the audiometer for self-calibration is most apparent. By addition of a built-in coupler phone or microphone with an appropriate coupling device and associated amplifier, VTVM or VU meter, instantaneous calibration of the system as a whole as well as many of its component parts can be effected. Such an addition to the standard audiometer would be most welcome.

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## CORRECTION

In the October issue of the ARCHIVES the article "Experiences with Transossicular Mobilization of the Stapes," by Dr. Mervin C. Myerson, was inadvertently placed in the Audiology Section instead of among the general articles.

Case Reports

# Diagnostic Problem of the Enlarged Parotid Gland

PHILIP H. SMITH, M.D., New York

Many times an otologist is called in on consultation in a general hospital to see a patient with an enlarged parotid gland. It is not unusual for a wide divergence of opinion as to the etiology. There is always the question of whether or not the enlargement is due to mumps; and, as luck would have it, the history of whether the patient had mumps is often confused. This situation arises whether the patient is a child or an adult. The purpose of this paper is to present a typical case and go through a differential diagnosis as an aid to future diagnosis and therapy.

## Report of Case

A man aged 36 presented himself for examination of swelling of his right parotid gland of 28-hours' duration. The gland was only slightly tender to palpation, symmetrically enlarged to about the size of a walnut, and firm to the touch. Stenson's duct orifice was minimally inflamed. No discharge could be expressed from the duct. The patient's temperature, complete blood count, and urine were normal. His only complaint was the enlarged parotid gland. No tumor or stone could be palpated, nor did the patient give any history of previous symptoms of parotid or duct stone. This was the first such episode of parotid enlargement. He further stated that his mother recalled vividly that as a child he had a "severe" case of mumps. The left parotid and the other salivary glands were normal. The eardrum membranes were normal. The spleen was not enlarged. There were no eye complaints.

The normal blood count ruled out blood dyscrasias, but Hodgkin's disease was a possibility. Mumps was ruled out by the history, as was calculus. A beginning tumor (benign or malignant) could conceivably cause tissue reaction and edema, and although none was felt, it might be too small to feel at this time. Uveoparotid fever was not considered because of lack of fever, uveitis, and such sundry symptoms as dryness of the mouth, facial paralysis, and burning of the tongue. The absence of lacrimal gland swelling and the normal spleen spoke against Mikulicz' disease. Pyogenic inflammation of the gland could hardly be present without pain and fever, and possibly chills. Inflammation of Stenson's duct with block by mucus plug or serous swelling would have produced pain and occasionally a history of recurrence. Tuberculosis was not considered.

At this point the diagnostic possibilities were Hodgkin's disease, beginning benign or malignant tumor, and viral inflammation of the parotid.

Therapy consisted of three daily injections of penicillin, 600,000 units each, and tetracycline (Achromycin) 250 mg. per os four times daily for three days. The patient was allowed to continue his normal activities. As is often the case in practice, the patient was reluctant to get x-rays of his lungs and parotid glands. On the second day the gland appeared to be somewhat smaller, and by the fifth day the area appeared normal. Cortisone was not given, nor was any local treatment applied. There has been no recurrence in 11 months.

## Comment

In retrospect, the patient must have had a viral inflammation of the parotid gland which responded to antibiotic therapy. Sometimes it is wise to defer elaborate testing and to use simple office tests until the direction the pathology is taking is clear. At that time specific testing is more logical.

## Summary

Painless swelling of the parotid gland presents a vexing problem. A case is presented with a differential diagnosis to illustrate methodology and therapy.

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1735 Townsend Ave. (53).

# Society Transactions

## CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Raymond W. Kerwin, M.D., President

Stanton A. Friedberg, M.D., Secretary

Monthly Meeting, March 4, 1957

**The Anatomy of the Descending Portion of the Facial Nerve.** DR. ANDREAS KODROS (by invitation), and DR. RICHARD A. BUCKINGHAM.

The authors presented the results of their observations in 100 temporal bones of the angle of descent of the third or descending portion of the seventh nerve.

They found that the third part of the nerve begins at the level of the lateral semicircular canal and descends, in 85% of the bones studied, at an angle of from 80 degrees to 90 degrees to the horizontal in the coronal plane. In 13%, the descent was from 70 degrees to 80 degrees, and in two specimens the nerve descended in a lateral direction of from 60 degrees to 70 degrees.

In the sagittal plane, the nerve descended forward from 100 degrees to 110 degrees in 9%, from 100 degrees to 80 degrees in 80%, and in 11% posterior to 70 degrees.

### Discussion

DR. SHERMAN L. SHAPIRO: I wish to ask Dr. Buckingham how his data compare with existing data on the variation in descent of the facial nerve?

DR. LEO A. SATZ: I should like to point out that variations in the course of the facial nerve in dangerous locations are usually mentioned as being present in the area where it makes its curve.

Danger of injury to the facial nerve is greatest at this location because the nerve comes so close to the structures of the posteroinferior tympanal walls even in a normal facial course, and that much more when the facial nerve varies in its course.

The tympanic sinus and the eminentia pyramidalis within the tympanic cavity lie only slightly inferiorly and anteriorly, respectively, to the curved section of the descending facial nerve. Many surgeons, when cleaning the tympanic sinus area of disease, have a tendency to go deep into the sinus and underneath the pyramidal eminence. Injury to the facial nerve at this point may occur, especially if sharp instruments are used and a little bit too much force.

For this reason, I feel that this excellent anatomical study of variations in the descending portion of the facial nerve has contributed greatly to our surgical knowledge.

Drs. Buckingham and Kodros deserve, therefore, a great deal of credit for their fine work.

DR. RICHARD A. BUCKINGHAM: Thank you very much, Drs. Satz and Shapiro.

I looked, and Dr. Kodros looked through the literature, and we could not find any description of the descent of the nerve. I am sure if we go back far enough we will find it all has been done, but we have not found anything as yet.

**The Larynx and Voice: The Function of the Normal Larynx (Motion Picture):** DR. HANS VON LEDEN, and DR. PAUL MOORE (by invitation).

The first in a series of educational motion pictures on "The Larynx and Voice," this film presents a brief review of the pertinent anatomy and a clear and detailed picture of normal laryngeal physiology in voice production.

The exact synchronization of action and sound gives an impression of realism and decisively resolves the controversial relation of the length of the vocal cords versus their mass in the determination of pitch.

The importance of ultra high-speed photography in the study of laryngeal function is demonstrated by a series of common sounds photographed at the rate of 5000 frames, or pictures, a second. The use of color film at such speeds represents a new development in laryngeal photography.

Newer observations on normal laryngeal physiology presented in this film include a new concept of abduction, arytenoid motion, and asynchrony of vibration. Similarly, the slow-motion pictures of the thick double lip of the vocal cords during phonation emphasize the importance of the loose mucous membranes, and contribute to a practical theory of voice production.

The complexity and diversity of laryngeal adjustments are illustrated by a cough and by a *coup de glotte*.

### Discussion

Dr. Albert H. Andrews Jr.: The opportunity of seeing this film is a thrilling one because it presents the dynamic picture of the vocal cords as seen with the routine use of the laryngeal stroboscope. This excellent film emphasizes the clinical value of stroboscopy in laryngology. The observation of the phonatory movements supplies a means of more precise laryngeal examination and evaluation of functional impairment.

I believe Drs. Von Leden and Moore and their group are to be complimented on their very excellent and very valuable work. All of us, I am sure, are going to learn a great deal from the present film and from the subsequent ones which will be helpful in our everyday practice.

Dr. Paul H. Holinger: First, I wish to comment on the film shown by Dr. Von Leden. Certainly this is an excellent film with beautiful photography and material so very well presented that it leaves hardly any room for comment. A discussion of the material presented in the film would take a separate evening. From the strictly physiological point of view, I think it might be well to review the arytenoid action which was suggested by an analysis of the film. I do want to compliment Dr. Von Leden on a most excellent presentation.

**Decannulating the Tracheotomized Patient with Poliomyelitis.** Dr. John Ballenger.

In general, there are only two principal physiologic considerations when decannulation of a case of bulbar poliomyelitis is contemplated: the ability to swallow effectively, and the presence of a "good enough" cough which, along with ciliary activity, can keep the lower respiratory tract clean. The first consideration is usually not difficult of determination. In an effort to make it easier to assess the quality of the cough, I shall review the dynamics of a normal cough with the idea that one or more of these factors can be used as a criterion of the effectiveness of a patient's cough.

Particular attention is paid to the function of the diaphragm in coughing. It is pointed out that the diaphragm is an antagonist of the anterior abdominal muscles which together provide the rigid floor of the thorax which is necessary for effective coughing. Loss of the abdominal muscles is the most important factor accounting for the pitifully poor coughs evident in patients recovering from poliomyelitis.

The following experiences are associated with 70 cases of poliomyelitis subjected to tracheotomy between 1950 and 1954 at the Evanston Hospital in Evanston, Ill. Vital capacity studies were related to predicted normal values. Among these are three patients in whom secondary tracheotomies were necessary following infection in the lower respiratory tract when it became evident that the cough was unable to keep the lower respiratory tract clean. It was concluded from these studies that the tracheotomy tube had been removed too soon.

Of the various factors which go to make up a normal cough, I found only two which held promise as a measure of the quality of the cough. The first of these is the vital capacity which has the disadvantage of being a static volume, whereas the act of coughing is a dynamic event. I conclude that decannulation should not be performed until the vital capacity has reached 30% to 40% of predicted normal. The second measure of a cough is the maximum expiratory pressure. Too little is known about this at present, but it may prove of value as additional information is obtained.

### Discussion

Dr. George A. Saxton Jr. (by invitation): It certainly has been a pleasure to be here this evening, not as an otolaryngologist, but rather as a respiratory physiologist.

In the treatment of poliomyelitis patients with respiratory paralysis during the past three and one-half years at the Respiratory Center of the University of Illinois, it has been our objective to get rid of the tube as soon as we felt it was feasible. Only patients with respiratory paralysis are admitted, about half of whom had tracheotomy tubes in place at the time of admission. We have used much the same criteria as Dr. Ballenger. Perhaps we are somewhat more inclined to get rid of the tube than Dr. Ballenger. This has been a trend in the care of poliomyelitis patients since 1951 or so.

Our criteria are as follows: 1. We have not removed any tube until the patient is able to swallow. 2. We do not remove the tube until we think that the tracheal secretions are manageable. By this we mean that the patient did not require suctioning more than once or twice a day. At this stage we often tape the tracheotomy tube closed and do not have to remove the tape to suction the patient more than once or twice a day. 3. We do not require any ability of the patient to cough spontaneously. All we require is that he be able to imitate a vacuum cleaner in reverse, as a blower, to inflate the lungs and cough during exhalation. This is not a very powerful cough because it depends chiefly on the elastic recoil of the lungs. However, it can be quite effective over a period of 10 to 20 minutes in Trendelenburg's position. We prefer this, as do the patients, to Barach's cough machine which forcefully inflates and exsufflates the lungs.

It is probably proper to say that we remove tracheotomy tubes as a calculated risk, usually in

three, four, or five months after the acute onset of the poliomyelitis. We now have 55 patients we have sent home, and none has ever been discharged with an open tracheostomy, although the vital capacity is very often 200 to 300 cc., and occasionally zero. We have had to reopen only one tracheostomy when a patient was readmitted because of an acute respiratory infection. However, we have had to reopen three while the patients were still in the hospital because of late complications three or more months after removal of the tube. Therefore, I think we would be inclined to continue with this somewhat more radical policy of removal of the tube.

Before concluding my comments, I would like to introduce to you this type of plastic tracheal plug. We use these as an intermediate device, between the time of pulling the tube out and letting the patient go home. After we have been able to tape the tracheotomy tube for a week or two, we will replace the taped silver tube with a solid plastic plug like this. There are different types of plugs being used. This one is shaped like a tracheotomy tube and projects into the airway. The flange at one end prevents the whole thing from slipping inside. There is a hole through one end outside the flange for a chain or string to go through and be tied around the neck. This one is made in California. Then there is another type made in Seattle which is a collar-button type; an outer cannula and an inner plug. It is shorter and, therefore, does not obstruct the air flow. We have not had as good luck with this type because it is somewhat painful to get this flange inside the trachea.

I must admit that at present we are facing the problem of a boy who has had four tracheotomies. His case is probably worth discussing because it illustrates some of the difficulties of decannulation. His first tracheotomy was performed within a few days of entering the hospital with acute polio and bulbar involvement. His physician at that time felt that the tube should not be left in too long, so they removed it 10 days after its insertion. It is not very often that a patient will be able to swallow and clear secretions that early, and he, I think, is representative of the group of patients who are decannulated too early. He then had his second tracheotomy as an emergency procedure four days later because of respiratory obstruction. He was transferred to our hospital with the tracheotomy tube in place two months later. We applied our criteria, and after a few months felt he was ready for decannulation. We took the calculated risk at about six months after onset, got the tube out, and sent him home. He acquired a respiratory tract infection and was readmitted to the hospital. Another tracheotomy was performed to suck out the tracheobronchial secretions. After his infection cleared, he was decannulated and discharged again. Recently, after being home for three to four months, he got another cold, returned to the hospital, and a fourth tracheotomy was done. I must say we feel somewhat discouraged in this case about the advisability of decannulation. However, as I said earlier, he is the only patient with whom we have had so much trouble. We now are planning to send him home with a plastic tracheotomy plug in the tracheostoma to keep it open in case he gets another cold. In the meantime, the portal of entry for infection will be somewhat closed and give him some protection. When we do send patients home with a plug like this, I think it is important to keep in mind the importance of suction apparatus. After a patient died here in Chicago last year because of a power failure, preventing use of an electric pump, we now supply a manual suction apparatus. This is somewhat less effective, but at least you can hope to keep the airway clear enough to prevent suffocation.

One final note about the usefulness of tracheotomy. In our experience, the cough machine of Barach has not been particularly helpful in raising secretions and opening atelectatic portions of the lungs. I think perhaps we have procrastinated too long sometimes before doing the tracheostomy, hoping the cough machine would suffice. However, deep tracheal suction has proved much more effective in clearing the tracheobronchial tree of secretions and reexpanding a collapsed lung. Therefore, I will close my remarks by praising the effectiveness of tracheotomy, though favoring the removal of the tube whenever possible.

DR. PAUL H. HOLINGER: In regard to the problem of removal of the tracheotomy tube, I must admit Dr. Saxton and I have discussed this and argued about it on a number of occasions. I was particularly interested in Dr. Ballenger's remark that, when the vital capacity of 30% is reached, decannulation can be considered. To use a mechanical value as an index of the time for decannulation I think would be an error; but to reach that point and then consider decannulation, I think might be an answer to this problem.

But what concerns us is why there is such a rush to decannulate these patients. We are more concerned about decannulation too early. The pediatricians, and perhaps Dr. Saxton, too, are more anxious to get the tube out than we usually are. We think that the tube serves a great purpose not only in facilitating aspiration of the bronchi at the moment, but also as a safety valve for the patient whose clinical history indicates he does have repeated respiratory infections and has to have help at intervals for aspiration of excessive mucus. We see these patients after decannulation and are asked to perform bronchoscopy when they get into trouble. Frequently we have to retracheo-

tomize the patient. It means rehospitalization for bronchoscopic aspiration when simple tracheotomy aspiration at home would have sufficed. Probably of greater importance is the damage caused by chronic failure of bronchopulmonary drainage. Lung damage does not take place in a moment or in a day or in a week, but takes place by repeated insults, repeated infections, repeated inability of the patient to get rid of secretions. What the patient eventually develops is the very serious problem of bronchiectasis. He will get over the acute respiratory infection; we know he will weather through it, but if he could rely on a little bit of help from the suction machine and the tracheotomy, I think he would avoid some of the lung complications.

I will agree that first and foremost the patient should have a well-fitting tracheotomy tube. An ordinary tracheotomy done without any regard as to whether the tube fits properly may result in great irritation to the trachea. A poorly fitting tube will increase mucus and cause much discomfort as well. A properly fitting tube is one which is checked with x-ray; it is of the proper length, and does not dig into the posterior or anterior walls of the trachea. With such a tube, the patient gets along reasonably well whether it is a plastic tube or something like the button Dr. Saxton showed us. If it is short and has the proper curve, whether it is made of silver does not make any difference; it must be a correct fit. It can be plugged when not needed for aspiration or respiration. When necessary, the tube can be opened to permit aspiration without taking the patient to the hospital for bronchoscopy. From our standpoint, we always say to the pediatrician who wants to get rid of the tracheotomy tube early, "Let's wait and see how he weathers his respiratory infection."

I wish to go back to Dr. Ballenger's statement, and say that we consider the time of removal of the tube to be later, rather than earlier, than Dr. Saxton would.

Dr. Thomas C. Galloway: The problem in poliomyelitis is to get the patient to live a more or less normal life without a tracheotomy, without the respirator, and I would agree with Dr. Saxton that we want to reach that ideal as much as we can.

In my experience, the patient who gets rid of the tracheotomy tube in the second, third, or fourth week is the patient who is much more likely to attain complete rehabilitation. We must remember that the patient in the respirator with a tracheotomy tube in place has surrendered control of his breathing through the Hering-Breuer reflex; he has surrendered glottic control; he has surrendered more than that—the will to cough, to breathe, and to swallow. The patient has to learn again to do these things. I think Dr. Ballenger has done a beautiful piece of work from the theoretical point of view, but I think it much more important to consider the patient practically. As Dr. Saxton said, if the patient can swallow his secretions, if his secretions are manageable, I think you would perhaps do better not to depend too much on the theoretical point of view. From my point of view, if the patient can swallow, if you can measure a reasonable movement of tidal air, and if the patient is comfortable, the tube should be removed. So many things can happen if you leave the tube in too long. One gets crusting and secondary infections, and the patient loses control of his own respiratory tract. The patient must live within his own powers. He cannot avoid infections, and there is excessive drying of the secretions from the tracheotomy. I think the patient who has rather early removal of the tracheotomy has by far the best chance of a normal life.

Unfortunately, Dr. Saxton gets most of his material rather late, and I think he has done a fine job with the cases he has handled.

Dr. Albert H. Andrews Jr.: The Barach exsufflation (inspiratory, positive pressure and high velocity, expiratory, negative pressure) has proved of therapeutic value in our experience. It is essential, however, that the apparatus be in perfect working condition, and this has required reconstruction of the valve mechanism. Its use in patients with retention of secretion complicating respiratory infections has been effective and may obviate repetitive tracheotomy.

Dr. Galloway's comment on the psychological surrender of the polio patient when placed in a respirator is pertinent. Consideration of assisted respiration, rather than controlled respiration, may be beneficial.

Dr. John J. Ballenger: I wish to thank those who discussed the paper, and particularly Dr. Saxton.

My last word is that I merely wish to say that we did have three cases who had to be retracheotomized; and one died before we were able to perform the operation. I agree that we should take the tube out as soon as it can be done safely, but you will have patients who will need retracheotomy to prevent death if the patient is decannulized too soon.

## CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

**Monthly Meeting, Feb. 4, 1957**

**Ossification of the Labyrinth in a Case of Glomus Jugulare Tumor.** DR. T. H. BAST.

During the past eight years many cases of glomus jugulare tumor have been recorded, but none in which there was also observed labyrinthitis ossificans. This latter condition was first described by Politzer in 1887 in a 13-year-old boy who, at the age of 2½ years, was severely ill with convulsions. Numerous similar cases were reported by others around the turn of the century and always with the history of severe infections of the cerebrospinal-meningeal type during the early years of life. In a case reported here, such ossification apparently did not occur until late in life. The patient died suddenly July 7, 1955, at the age of 60 years, two days after a second mastoidectomy.

The main entries on the patient's medical record show tonsillectomy in 1929, uterine fibroids with hysterectomy in 1930, and stilbestrol medication since the latter date. Her ears became stopped up in 1932. Hearing loss and pounding tinnitus in left ear gradually increased between 1937 and 1945. In 1947 a husky voice was noted. Her tongue became thicker; there was difficulty in swallowing, and a serous discharge from the left ear. The patient veered to the left in walking. A polyp was removed from the left ear in 1949. She entered Wisconsin General Hospital for the first time in February, 1948, and was transferred to the Otolaryngology Department in May, 1948, where the initial impression was middle-ear disease with polyp. X-ray showed active infection in the left mastoid. Basal skull films were negative. A very vascular polyp was removed from the left ear. Radioactive phosphorus ($P_{32}$) was given with a resulting decrease in size of the mass for a short time, but there was no change in the mastoid. Radical mastoidectomy was done June 15, 1949, and as much of the friable vascular tumor mass removed as was possible. It had invaded the floor of the middle ear from the jugular bulb region. The condition remained quiescent until December, 1954, when a large swelling appeared on the left side of the neck together with reappearance of the tumor of the left middle ear. There was neurological evidence of involvement of 5th to 12th cranial nerves. On Jan. 3, 1955, a neck dissection was done with partial removal of a tumor mass involving the left jugular vein. The diagnosis was glomus jugular tumor. X-ray therapy consisting of 3000 r was given in 12 treatments. The tumor in the left ear continued to enlarge, and vestibular involvement became more marked. A second and final mastoid exploratory operation was performed July 5, 1955, and the patient died two days later.

At autopsy the left petrous bone was removed, prepared, and sectioned in series. The tumor had invaded all petrous air cells and even the bone marrow of the petrous apex. The interesting fact, however, was that all portals of entry to the labyrinth, viz., secondary tympanic membrane, aquaeductus chochleae, aquaeductus vestibuli, and internal acoustic meatus, were resistant to the tumor, and no tumor tissue was found anywhere in the labyrinth. Instead, the entire labyrinth was filled with new bone with the exception of a small part of the vestibule. This labyrinthine ossification apparently developed independently from the glomus tumor.

The cause of this ossification is not known. The sections did show that the stapes was dislocated along its anterior margin, probably owing to middle-ear packing following the tumor removal. A dense, well-developed but vascular connective tissue filled the gap between the oval window and the displaced part of the stapes. It is possible that an infection could have entered the labyrinth via this ruptured annular ligament and that this infection may have initiated the ossification. The only objection to this etiology is the fact that the new bone which fills the labyrinth did not invade the area where the stapes ligament was ruptured. Thus, the origin of the ossification remains unknown. In the future it will be of interest if labyrinthitis ossificans will be noted in cases of otitis media following dislocation of the stapes in attempted stapes mobilization.

### Discussion

DR. JOHN R. LINDSAY: When I was asked to discuss this case, the two thoughts which occurred to me were whether I should say something about ossification of the labyrinth, which is, I think, Dr. Bast's main interest in this case, or whether I should discuss the clinical aspects of glomus tumors.

This case which Dr. Bast has presented reminds me of a case we treated before the days of penicillin which had a similar, long history of previous operations, a facial paralysis of several years' duration, and a dead ear. We thought we were dealing only with a suppurative problem, but on operation we found we were dealing not only with granulation but with tumor tissue. The patient died from meningitis. The histological study of the ears showed not only labyrinthitis ossificans such as was

found in Dr. Bast's case, but also a subluxation of the stapes, and I feel that it is likely that the same thing occurred at the previous operation in Dr. Bast's case. In controlling the severe bleeding with packing, the stapes may easily be subluxated.

The labyrinthitis was the result of infection of the labyrinth rather than tumor.

We have examined several cases of tumor of the temporal bone, where the tumor literally surrounded the capsule, but it is surprising how resistant the endosteum is to invasion by the tumor. It seems that this dense, almost avascular bone, can be extensively eroded before the tumor is able to invade the labyrinth. Then, again, we have only a few cases where the labyrinthitis ossificans resulted from invasion by infection in the petrous pyramid. Many of the cases of labyrinthitis ossificans reported in the literature were due to meningococcic meningitis from which the patient had recovered but in which a dead labyrinth had developed. I am of the opinion that ossification of the labyrinth in this case came about as a result of tympanogenic labyrinthitis.

A question of primary concern to us is the treatment of glomus tumors. I have one case under observation at present whose symptoms began 15 years ago. This patient developed a paralysis and atrophy of one side of the tongue, paresis of the 11th nerve, paresis of one side of the pharynx, and also a dead ear. The sixth and seventh nerves are also affected. This patient is a cripple because she cannot swallow properly and has poor articulation. There are extensive changes shown on x-ray. In 1954 she was treated by radiation therapy. I saw her recently, and she looked better than she did three years ago. However, she is still a cripple. This case illustrates the slow rate of progression of these tumors.

On the other hand, in two of four cases I operated on (three within the last few months) it was possible to save even the ear drum and ossicular chain by removing the bone of the posterior and inferior canal walls, and then lifting the ear drum out of its sulcus. The tumor had not become fixed to the ear drum, and after removal the drum was replaced. These were cases where the tumor apparently arose from the promontory (Jacobson's nerve), whereas in other cases it may arise from the bulb. In such early cases surgery certainly seems to be the treatment of choice. One of these early cases was treated by x-ray before surgery but showed no change in appearance and no relief of tinnitus, so I removed the tumor. I operated on another case about three years ago with complete facial paralysis and symptoms of about four years' duration. The slides of Dr. Bast reminded me of that case because every mastoid cell bled furiously at operation, so I am now sure there was tumor all through those mastoid cells. I saw that case a year later, and there was a nice result. The cavity was healed, and facial function had partially returned. I have not seen the patient for a year and after seeing these slides, I doubt that he is cured, but he was greatly improved.

My opinion is that when the ear is functioning and destruction is not so extensive as to preclude removal with reasonable safety, these cases should be treated by surgery.

Dr. Williams gave a paper at the Otological Society a few years ago reporting very good results from x-ray therapy in this type of tumor. My own opinion is that we ought to use x-ray in advanced cases where operation is not feasible. In the less advanced cases, particularly those which arise from the promontory or in the middle ear, surgery is the treatment of choice. Granted we may not be sure of a cure, but if there should be a recurrence, it is likely to be recognizable and can be more easily dealt with.

**Principles of the Use of Steroids in Otolaryngology.** DR. RACHMIEL LEVINE (by invitation).

The program listing is somewhat erroneous in stating that I would speak of the use of the steroids in otolaryngology. I think it would be more correct to say that I will talk on the principles of the use of the steroids, and leave out otolaryngology, since I know practically nothing of the field except that I am acquainted with some practitioners in that specialty.

The word "steroids" in this context refers only to a few substances out of the very large group designated as steroids. The steroids referred to all have an oxygen atom at carbon[11] and at carbon[17], and they are designated as the 11-17 oxysteroids. Those are the substances that are in use for the alleviation of symptoms and signs in the various branches of medicine.

The adrenal cortex secretes three types of steroids. One is the type similar to desoxycorticosterone, the main action of which is to retain water and salt when the steroid is given to the organism in vivo. This group of steroids is of no particular value or interest in the field we are exploring tonight.

The second group are the androgens secreted by the adrenal of both sexes. These androgenic substances also do not have many of the properties of the steroids we are interested in.

The adrenal cortex in addition secretes substances of the third type, which are the 11-17 oxysteroids.

There is absolutely no relation between the function of the adrenal cortex as an endocrine gland and the use of the steroids in medicine. The use of these steroids is purely pharmacological, and the analogy which comes to mind right now is the use of insulin shock therapy in schizophrenia. Insulin shock therapy is not given because of an insulin deficiency. None of the disorders commonly treated with steroids, except for the diseases of the adrenal itself, have any relation to the function of the adrenal cortex. There is really no logical scientific basis for the widespread use of the steroids in medicine. If an endocrinologist had been asked in April, 1949, whether, on the basis of his knowledge of the function and physiological effects of the 11-17 steroids, he could deduce their eventual use in any nonadrenal disease, he would never have predicted their use as anti-inflammatory and anti-allergic agents.

We do have, however, a certain body of knowledge that has been gathered since May, 1949, as a result of the empirical alleviation of the symptoms of rheumatoid arthritis, which now gives us a background and a set of guiding principles as to the use of steroids in disease. We have also gathered a certain amount of knowledge about the structure of these compounds in relation to their therapeutic effects. Modifications from the original cortisone and hydrocortisone have appeared and been found helpful.

Soon after the discovery of Hench and his associates, the use of these compounds was extended to disorders which resembled rheumatoid arthritis or rheumatic fever, the collagen diseases. From this vantage point the use spread to the allergic disorders. Experimental observations demonstrated that a common feature of the action of cortisone was its anti-inflammatory effect. It suppressed granulation tissue proliferation in a variety of induced inflammations. This action is exerted locally in the inflamed site, since one can inject a small amount of cortisone with the irritant into the same area and inhibition of inflammation will occur. In general, one only has to use a very small amount of the substance when it is injected directly into the injured area without affecting the animal as a whole. Parallel with these animal investigations were the observations in the human. Surface inflammations such as conjunctivitis could be treated locally; so could single rheumatoid joints. It is apparent that the effect of cortisone is exerted directly in the area of inflammatory reaction. Whatever the inflammatory stimulus, the normal reaction proceeds in a spherical manner from the site of the irritation. If cortisone is also present, the cells cannot react to the irritant. This was demonstrated in 1951 and 1952 by many experiments, of which the following is an example. It is known that one can produce experimental nephritis by injecting antibodies in an especially prepared animal. The antigen-antibody combination occurs in the basement membrane of the glomerulus and serves as an inflammatory stimulus to the tissue around it. If the animal is pretreated with cortisone, the course of the disease is altered. There is no evidence in the urine, no evidence in the behavior of the animal, and no evidence on pathologic section of the development of glomerulonephritis. When cortisone administration is interrupted, the glomerulonephritis develops as if the antibody were given at this time. As soon as the protective effect is removed, the inflammation occurs.

In rheumatoid arthritis, when steroid medication is stopped, flare-up of the rheumatoid process may occur. The steroids are in no sense curative.

The steroids are anti-inflammatory; their effect does not seem to depend on the nature of the inflammatory agent; they act locally; they do not represent anti-etiologic therapy; they simply relieve symptoms and signs associated with a simple or complex allergic or inflammatory reaction.

The steroids have, of course, actions other than anti-inflammatory. These have wrongly been called side-effects. They are really the exaggerated hormonal activities of these substances. Protein breakdown is hastened which, on long-standing administration, may lead to dangerous osteoporosis with fractures, especially of the vertebral column. The formation of sugar from protein is increased, and this may predispose, in about 2% of the cases, to hyperglycemia. Sodium and water are retained, and there is an increase in the excretion of potassium. Thereby hypertension and edema may be produced. The skin thins and a redistribution of the fat occurs. This is the induced Cushing's syndrome seen formerly only in adrenal hyperplasia or tumor. These effects are inevitable whenever cortisone or one of its cousins has to be used in large dosage for long periods of time, such as in severe cases of lupus, pemphigus, periarteritis, and in some cases of very severe inflammatory rheumatism. Below certain dosage levels these complications need not occur, (usually below 50 mg. of cortisone or its equivalent). Another serious complication is of special interest to surgeons. Whenever any of the active steroids are administered, they raise the level of these materials in the blood. This serves to depress corticotropin (ACTH) secretion by the pituitary. The patient's adrenals begin to diminish in size, and may become atrophic. Fortunately, in the overwhelming majority of instances, the process is reversible as soon as the administration of these drugs is stopped. However, when a patient has been under the influence of outside cortisone for too long, one should not remove medication abruptly, because the atrophic state of the adrenals may lead to an Addisonian

crisis. When an additional strain is imposed on the patient, he may die in peripheral vascular collapse of the adrenal variety. One can overcome this by withdrawing the drug very gradually. This gives the pituitary a chance to increase its own activity and, in turn, bring the adrenals back to normal. There have been publications which can be interpreted to mean that in some instances such adrenal depressions are not reversible. I have not had that experience.

One of the ways to obviate the above difficulty is to give the cortical hormone by mouth every day and then periodically (every week or every two or three weeks) to inject ACTH. There is another way of obviating difficulty. It is possible sometimes to gain access to inflammatory or allergic processes without having to give parenteral medication. In dermatology, in otolaryngology, and certain other fields, the local application of the potent cortical hormones is being utilized, which has an excellent rational basis.

Now something about the substances themselves. Cortisone and hydrocortisone were the first compounds used. The ratio of activity, if cortisone is 100%, is 125 for hydrocortisone or about 25% more potent per mg. Of these, hydrocortisone is a normal hormone of the adrenal cortex. Prednisone (Meticorten) and prednisolone (Meticortelone) are from four to five times as potent as the standard, per milligram. That by itself has no advantage; it doesn't matter at all whether something is more potent per milligram. The number of complications is about the same, but there is one advantage of Meticorten and Meticortelone, and that is that the retention of sodium and water is much less in therapeutic doses than it is with cortisone or hydrocortisone. There is a clinical suspicion that bleeding peptic ulcers are commoner with prednisone and prednisolone than with the original cortisone and hydrocortisone.

While the steroids prevent inflammatory reaction and pain, they at the same time prevent the formation of the barrier around the inflammatory focus. Therefore, viral and bacterial spread may occur. In the presence of any focus of infection the antibiotics have to be used simultaneously with the steroids. This same type of situation is probably the cause of the bleeding peptic ulcer. Peptic ulcer is an inflammatory nidus and has a barrier, but when steroids are given, two things happen. First, the acid-secreting activity of the stomach is enhanced, and, second, the healing process will be inhibited.

The steroids have been in some instances life-saving; in many more instances they have prevented pain and disability. However, they have also produced complications which we had not foreseen. The cortical substances are useful if intelligently handled, but they should not be dispensed as if they were harmless analgesics.

## Discussion

Dr. Francis L. Lederer: I am grateful for the opportunity of listening to this splendid presentation on steroids. I am sure that Dr. Levine has not made many friends tonight because he has failed to tell you that this is the "cure-all" to the many ills that befall our otolaryngological mankind. We are aware that since observations were made on the empirical bases referred to, there has been a good deal of abuse of their application. I suppose the next paper may be on the abuse of the steroids in otolaryngology. Each time we do not know what to do next, and when aspirin or antibiotics have not come to the rescue of the patient, we use steroids. The detail salesman is our best bet.

Today we are residing in a generation that, when we do not know something, we give cortisone. In desperation we have been using these preparations in the treatment of nasal polyps. We have been overwhelmed and delighted to find that polyps presenting themselves in the anterior nares would disappear with this therapy. The patient, of course, was so grateful he wanted to keep on taking these agents particularly when, if they were discontinued, the polyps recurred. I am glad to be enlightened about the use of the steroids and the terrible "side-effects" which now must be called complications. We have in the past been faced with disagreeable complications, particularly infections, because there was no resistance on the part of the body structure. Tonight we see just why this has occurred.

Now, surely, after listening to this comprehensive presentation, we will hesitate before we prescribe this type of remedial agent.

I was interested in the fact that in his early remarks, Dr. Levine mentioned that the emotions had something to do with the way these agents acted. We formerly heard of patients who were given this remedy for rheumatoid arthritis, for example, who would become so euphoric as to practically be climbing the ceiling. I want to ask Dr. Levine about this, whether this is still to be reckoned with as a complication, or whether this has been overcome.

I wish to express my pleasure at the opportunity of not only meeting Dr. Levine, but in listening to this wonderful presentation.

Dr. Phillip Mozer (by invitation): Dr. Levine, I wish to ask what you feel are the prospects for the topical application of the cortisone substances. Is there absorption after prolonged use?

Dr. Rachmiel Levine (by invitation): It depends on the amount and type you use. Fludrocortisone is a very potent agent. It is practically

impossible to give it parenterally since it is a powerful sodium retainer. Topically it is quite useful. One of the advantages of Fludrocortisone is that one can gauge the amount absorbed and keep it under the amount which will produce any systemic symptoms. Usually cortisone, by whatever route, is absorbed. One of the interesting things is that it is usually metabolized or excreted in 12 to 24 hours, so that there is no cumulative effect. One might give 100 mg. of hydrocortisone, and suppose this were all retained for a day and not excreted or metabolized but distributed throughout the total body water. In an adult the total body water is about 50 liters, so the concentration would be 100 mg. in 50 liters, or 100 in 50,000 ml., or 0.2 mg. per 100 cc. If one puts on topically 0.5 mg. on the particular area, you are getting probably 10 times the amount you actually need. That is why it is better if you can get a little bit where you want it. In a systemic disease there is the difference that one cannot apply a little bit over the lesions of lupus, for instance.

Dr. Lederer mentioned the complication of psychosis from therapy with steroids. This is not as threatening or the damage as great as we used to think it would be, during the first six months of 1949, or in 1950. It occurs with much less frequency than I would have expected. On the other hand, peptic ulcer became apparent about 1950, and hemorrhage from this is now a more frequent complication.

Dr. Gail Soper (by invitation): What would be the care in cases on whom you are going to do tonsillectomies, if they have been on cortisone therapy for more than two weeks and are on it at present?

Dr. Levine: Do you mean the patient is getting cortisone at the time of surgery?

Dr. Soper: Yes. I wondered what one would do in that case.

Dr. Levine: If the patient is getting cortisone, continue it. The normal adrenal puts out in 24 hours somewhere between 12 and 24 mg. of cortisone. All one needs, in the complete absence of adrenal function, is such an amount. Under stress, the normal adrenal secretes five times this amount. If anyone, therefore, is receiving something between 60 and 120 mg. of cortisone or the equivalent for some systemic disorder and is being operated on, there is no need for additional cortisone. This person has as much as the normal adrenal will be able to put out under the most stressful conditions. If, however, the patient has had cortisone which has been discontinued and two weeks later has to undergo operation, then one should give 100 mg. of cortisone or the equivalent daily for several days, and then decrease it slowly.

Dr. Lederer: What about the factor of surgery and infection under those same circumstances?

Dr. Levine: Infection is always a hazard. Whenever you fear infection, you have to use some of the broad-spectrum antibiotics. If you have a tuberculous patient, particularly, there are the more specific antibiotics. Sometimes cortisone is used in infections. For example, in tuberculous meningitis in which there are many fibrous adhesions, the antibiotics may not be able to get to them too effectively, and cortisone will inhibit the formation of fibrous adhesions and may lead to lysis. Therefore, cortisone is given with a high level of streptomycin and other antituberculous agents, in order to overcome such infections.

Dr. Arthur Ratko: Does the topical application of these substances have an effect on the healing process?

Dr. Levine: On the healing of wounds?

Dr. Ratko: Yes. I wondered about the healing of wounds by first intention. Is there any explanation for the different action in wound healing? Can you explain why the normal expected response does not take place? The healing process, after all, is merely a modified form of an inflammatory process. Why should there be a difference between the alteration of the inflammatory response and the apparent failure to affect healing?

Dr. Levine: I do not know. It is quite possible that if you took cortisone or Fludrocortisone and put it on the edges of the wound it might interfere with its healing, but I doubt that the concentration it gets would be high enough to interfere with the normal stimulus to repair. It seems to be a somewhat different process, for example, than in inflammation of infection.

Dr. Burton Soboroff: Is there any evidence to show that the final fibrosis and scarring is a chemical reaction, and does this make it any less, more, or just as likely with the use of the cortisone substances?

Dr. Levine: This is very difficult to state because it is not really known. Cortisone has been used, for example, in keloids. Some have reported benefit; other workers have not seen any change. Cortisone has been given to prevent adhesions in the peritoneal cavity to aid in certain aspects of gastrointestinal surgery, but in these areas it is still open to investigation, and there are no definite data available.

Dr. Arthur Loewy: The steroid substances for topical use are available in strengths of from 0.5% to 2.5%. Is there a proportionate difference in their local action, or is there a maximum topical strength beyond which any increase is not any more clinically effective?

Dr. Levine: I think that would depend on the extent of the reaction and how near you can get

to it with the material. This is purely speculation, because I do not think it is known. There may be an error of 100% up or down.

Dr. Robert Henner: Why has there been sensitivity to the mycins when used in combination with cortisone? Is there a technical difference in the sensitivity reaction to the mycins?

Dr. Levine: There are certain inflammatory and allergic reactions where cortisone will have an effect. In the anaphylactoid reactions cortisone does well. On the other hand, in Arthus' phenomenon the ulceration does not seem to be prevented. So there is a difference between these inflammatory reactions.

Dr. John R. Lindsay: In fibrous tissue formation in case of burns of the esophagus, is there any rationale to the use of cortisone?

Dr. Levine: Yes, there is some rationale to that. I would say if steroids are given very early, immediately after the burn, during the time there is serous exudation and edema, there might be benefit. I doubt very much if cortisone would help after the fibrous tissue had formed.

Dr. Lindsay: Could local applications be used, or would parenteral administration be best?

Dr. Levine: It would seem that you would have to use large parenteral doses because the local reaction would be a diffuse one.

# News and Comment

## ANNOUNCEMENTS

**American Association for Cleft Palate Rehabilitation.**—The 10th Annual Convention of the American Association for Cleft Palate Rehabilitation will be held at the St. Francis Hotel in San Francisco on Thursday, Friday, and Saturday, April 24, 25, and 26, 1958.

This association is composed of medical, dental, and paramedical specialists who are interested in the rehabilitation of persons with cleft lips and palates. D. C. Spriestersbach, Ph.D., Department of Otolaryngology, University Hospitals, Iowa City, is Secretary-Treasurer.

## GENERAL NEWS

**International Voice Conference.**—The International Voice Conference, dealing with laryngeal research, physiology, and therapy, was held May 20-22, 1957, in Thorne Hall on the Chicago campus of Northwestern University, under the joint chairmanship of Hans von Leden, M.D., and Paul Moore, Ph.D., with the cooperation of representatives from all Universities in the Chicago area.

Delegates from 25 foreign countries and the United States represented the leading laryngeal research centers. Thirty-eight scientific papers covered all aspects of laryngeal research. The Guest of Honor, Dr. Eelco Huizinga, Professor of Otolaryngology at the University of Groningen in The Netherlands, addressed the formal closing ceremonies on the subject: "The Larynx and Voice—Animals and Man."

At the same meeting Mr. W. F. Gould, President of the William and Harriet Gould Foundation, announced an annual award for outstanding research in the field of laryngology. First recipient of this award was Dr. K. Faaborg-Andersen of Copenhagen, for his electromyographic studies of internal laryngeal muscles in humans.

# Books

## BOOKS RECEIVED

* **Magnetic Removal of Foreign Bodies.** The Use of the Alnico Magnet in the Recovery of Foreign Bodies from the Air Passages, the Esophagus, Stomach and Duodenum. By Murdock Equen, M.D., F.A.C.S. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill. Price, $4.50. Pp. 94, with 119 illustrations. Published simultaneously in the British Commonwealth of Nations by Blackwell Scientific Publications, Ltd., Oxford, England, and in Canada by The Ryerson Press, Toronto, 1957.

**Ciba Foundation Colloquia on Endocrinology, Volume X: Regulation and Mode of Action of Thyroid Hormones.** Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Elaine C. P. Millar, A.H.-W.C., A.R.I.C. Price, $8.50. Pp. 311, with 114 illustrations. Medical Book Department, Little, Brown & Company, 34 Beacon St, Boston 6. Published in England by J. & A. Churchill, Ltd., 104 Gloucester Place, London W. 1., 1957.

**Ciba Foundation Symposium: The Chemistry and Biology of Purines.** Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Cecilia M. O'Connor, B.Sc. Price, $9.00. Pp. 327, with 124 illustrations. Medical Book Department, Little, Brown, & Company, 34 Beacon St., Boston 6. Published in England by J. & A. Churchill, Ltd., 104 Gloucester Place, London W. 1., 1957.

* **Practical Otolaryngology.** By Gervais Ward McAuliffe, M.D., F.A.C.S., F.I.C.S. Price, $7.00. Pp. 320, with 29 illustrations. McGraw-Hill Book Company, Inc., 330 W. 42nd St., New York 36, 1957.

* **The Rehabilitation of Speech:** A Textbook of Diagnostic and Corrective Procedures Based upon a Critical Study of Speech Disorders. Third Edition. By Robert West, Merle Ansberry, and Anna Carr. Price, $7.50. Pp. 676, with illustrations, charts, tables, glossary. Harper & Brothers, 49 E. 33rd St., New York 16, 1957.

* **Diseases of the Nose, Throat and Ear.** Tenth Edition. By Howard Charles Ballenger, M.D., F.A.C.S., and John Jacob Ballenger, B.S., M.S., M.D. Price, $17.50. Pp. 968, with 550 illustrations and 11 color plates. Lea & Febiger, 600 S. Washington Square, Philadelphia 6, 1957.

* **The Early Diagnosis and Treatment of Acoustic Nerve Tumors.** By J. Lawrence Pool, M.D., and Arthur A. Pava, M.D. Price, $5.50. Pp. 161, with illustrations, charts, tables. Charles C Thomas, Publisher, 301-327 E. Lawrence Ave., Springfield, Ill., 1957.

* **Oral Medicine.** Third Edition. By Lester W. Burket, A.B., D.D.S., M.D., Sc.D., with a chapter on Oral Cancer by S. Gordon Castigliano, B.A., B.S., M.D., F.A.C.S. Price, $14.00. Pp. 558, with 391 illustrations (including 40 subjects in color). Medical Department, J. B. Lippincott Company, E. Washington Sq., Philadelphia 5, 1957.

* **Topographische Ausdeutung der Bronchien im Röntgenbild unter besonderer Berücksichtigung des Raumfaktors.** By Priv.-Doz. Dr. Med. Claus Esser. Price, $13.80. Plates 89, with 157 individual illustrations. Georg Thieme Verlag, Herdweg 63, (14a) Stuttgart N, Germany, (In the U. S. A. and Canada: Intercontinental Medical Book Corporation, New York 16), 1957.

* **Der Hirnabszess.** By Priv.-Doz. Dr. Gerhard Weber. Price, $7.10 (Paper-back edition). Pp. 185; charts and tables (not numbered). Georg Thieme Verlag, Herdweg 63, (14a) Stuttgart N, Germany. (In the U. S. A. and Canada: Intercontinental Medical Book Corporation, New York 16), 1957.

* To be reviewed.

# Directory of Otolaryngological Societies *

## FOREIGN AND INTERNATIONAL

### Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.
Place: Nova Scotian Hotel, Halifax, N. S. Time: June 9-11, 1958.

### Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

### International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh International Congress of Bronchoesophagology.
Place: Kyoto, Japan. Time: Sept. 14-16, 1958.

### International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. Henry M. Scheer, 522 W. End Ave., New York 24.
Secretary: Dr. Louis Savitt, 3402 W. Lawrence Ave., Chicago 25.

### International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

### International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

### Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Brazil. Time: Aug. 12, 1958.

## NATIONAL

### American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Alden Miller, 500 S. Lucas Ave., Los Angeles.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco. Time: June 23-27, 1958.

### American Academy of Ophthalmology and Otolaryngology

President: Dr. Erling W. Hansen, 90 S. Ninth St., Minneapolis.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

### American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

### American Broncho-Esophagological Association

President: Dr. Walter Hoover, 605 Commonwealth Building, Boston.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

### American Laryngological Association

President: Dr. Harry P. Schenk, 326 S. 19th St., Philadelphia 3.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: Fairmont Hotel, San Francisco. Time: May 19-20, 1958.

### American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

SECTIONS:

Eastern.—Chairman: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Place: Hotel Warwick, Philadelphia. Time: Jan. 9-10, 1958.
Southern.—Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Peabody Hotel, Memphis, Tenn. Time: Jan. 25-26, 1958.
Middle.—Chairman: Dr. Francis L. Lederer, 860 N. Lake Shore Drive, Chicago.
Place: Hotel Morrison, Chicago. Time: Jan. 13-14, 1958.
Western.—Chairman: Dr. Ben. R. Dysart, 960 E. Green St., Pasadena 1, Calif.
Place: Beverly-Hilton Hotel, Beverly Hills, Calif. Time: Jan. 18-19, 1958.

### American Rhinologic Society

President: Dr. Walter E. Loch, 9 Beechdale Rd., Baltimore 10.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.

### American Otological Society, Inc.

President: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: Fairmont Hotel, San Francisco. Time: May 17-18, 1958.
Place: The Homestead, Hot Springs, Va. Time: 1959.

### American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

### American Society of Facial Plastic Surgery

President: Dr. Irvin J. Fine, 505 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.

### American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2.

### Otosclerosis Study Group

President: Dr. Joseph A. Sullivan, 174 St. George St., Toronto 5, Canada.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.

### Society of Military Otolaryngologists

President: Capt. William C. Livingood, U. S. N. (MC).
Secretary-Treasurer: Lt. Col. Stanley H. Bear, USAF(MC), 3810th USAF Hospital, Maxwell AFB, Alabama.

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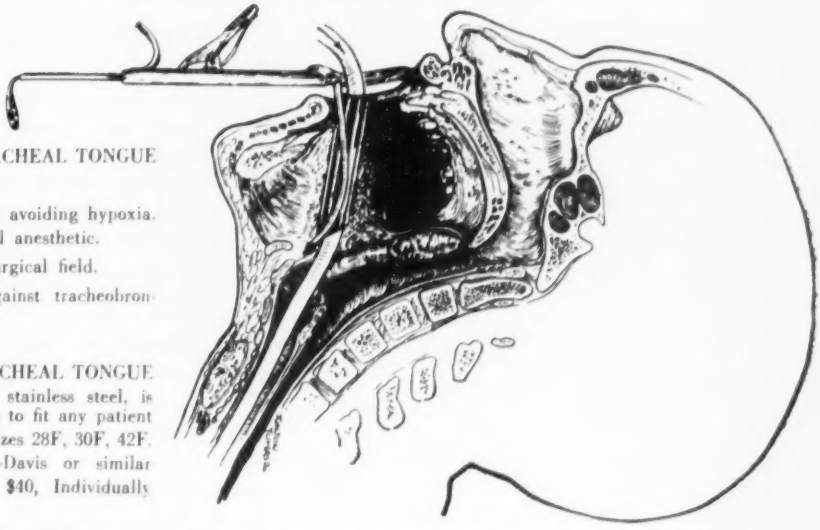

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*Barton, R. T.: Instrument for endotracheal anesthesia during oro-pharyngeal surgery. J. A. M. A. 153:1017 (Nov. 14) 1953.

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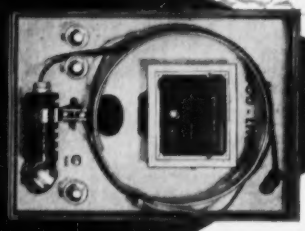

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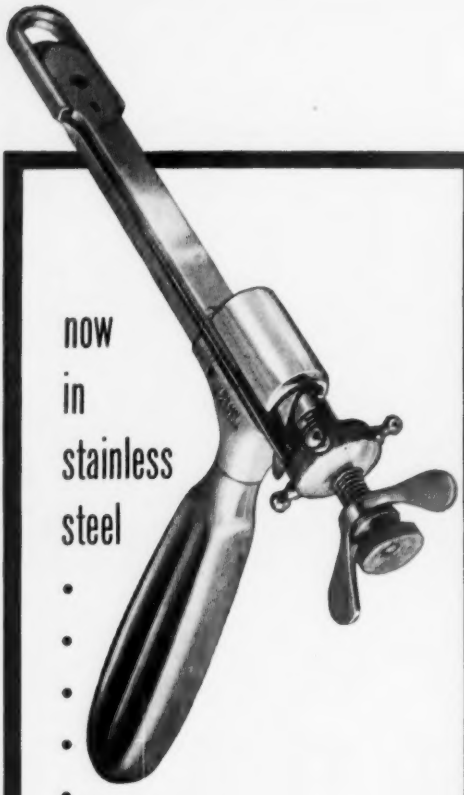

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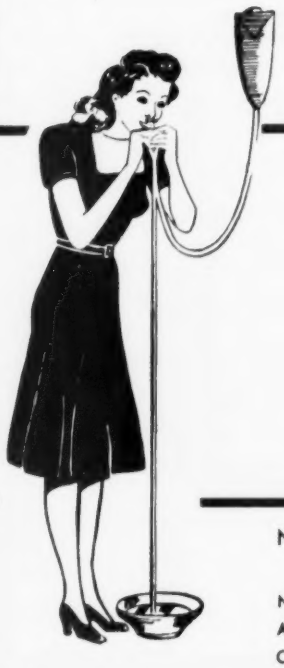

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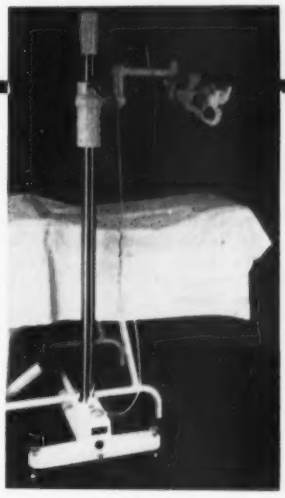

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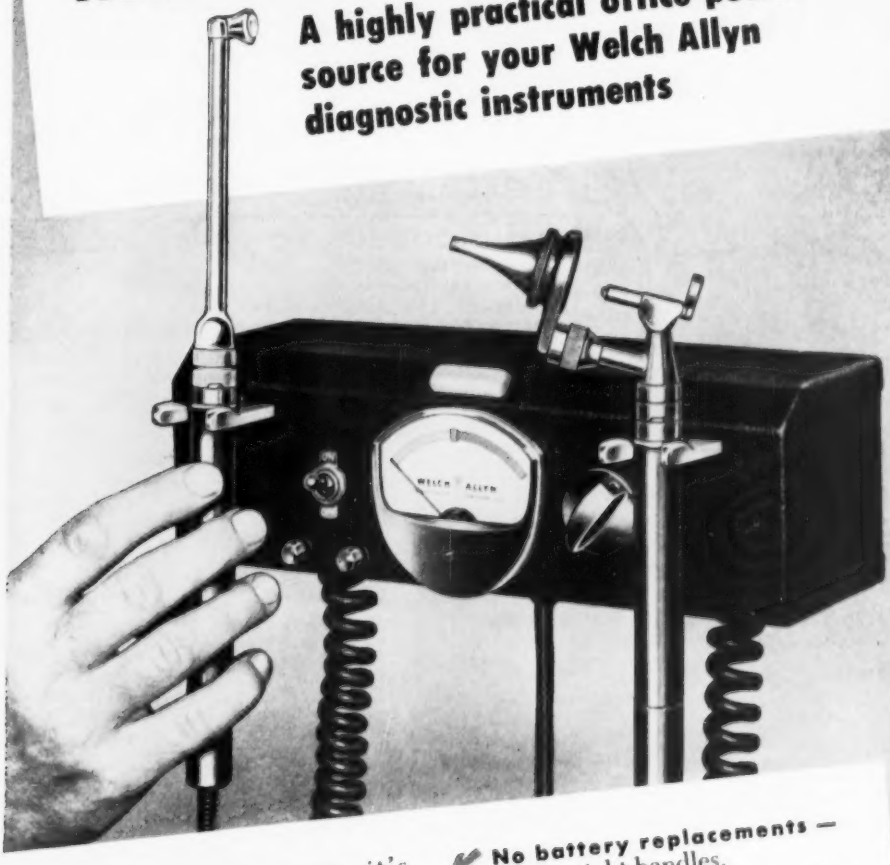

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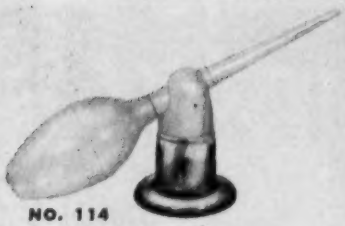

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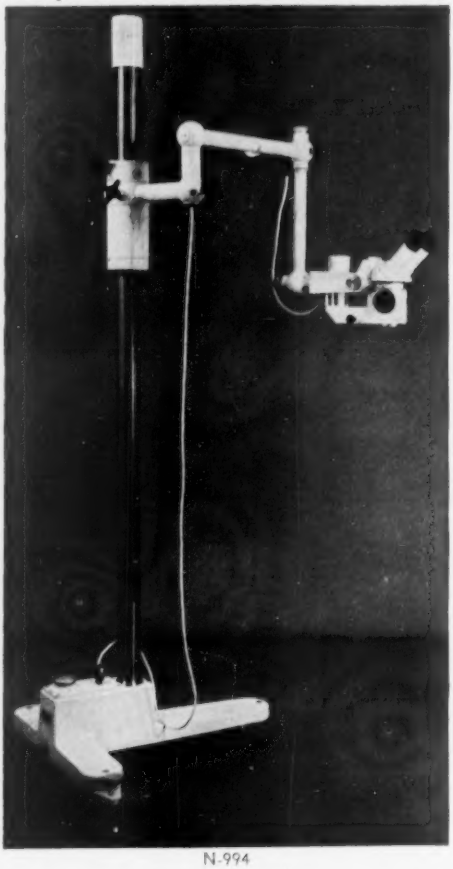

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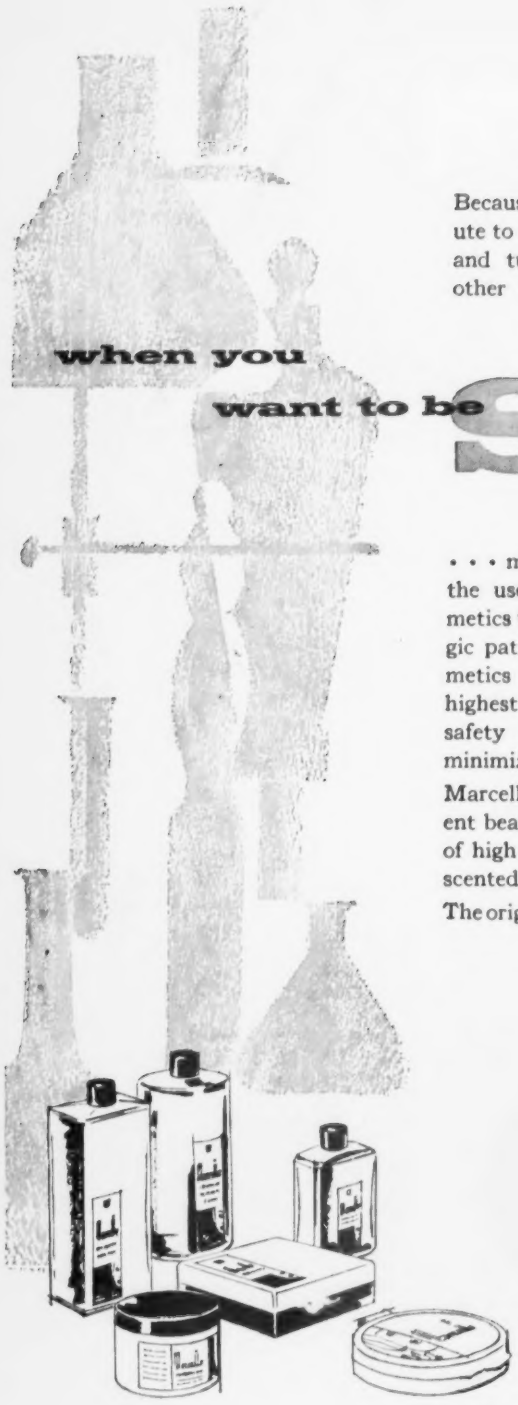

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